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ANALYSES OF THE ASH OF THE NORMAL AND THE CATARACTOUS LENS.

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THE following investigation forms a part of a larger series of analyses of the ash of different organs undertaken principally to ascertain what changes, if any, occur in the inorganic constituents of the tissues as a result of old age. Quite early in the work it became possible to get a large supply of human cataractous lenses in their capsules and it was thought desirable to study the changes that occur in the ash of the lens under these conditions. For the supply of cataractous lenses I desire to thank especially Dr. D. W. Green of Dayton, Ohio, who has been most generous in sending on many hundred of lenses exhibiting senile cataract and in many cases furnishing also the clinical histories of the cases. Through the kindness of Drs. Hugh Young and James Bordley, I was put in possession of a second large supply of similar lenses which had been collected in India by Dr. Smith. The abundant material thus obtained has facilitated greatly the work of obtaining satisfactory analyses, since certain difficulties were encountered, especially in the determination of the sodium, which required frequent repetitions of the analytical work. On the other hand, great difficulty was experienced in obtaining normal human lenses. The comparatively scanty material of this kind that it was possible to collect was obtained from time to time from autopsies. It was not, however, entirely adequate for the purposes of analyses and I

was obliged to make use of the pig's lens and many hundreds of the latter were used in the work, and the results from this source constitute the more reliable data as regards the ash of the young normal lens. It will be observed upon examination of the tables that the percentage of ash of the normal pig's lens is practically identical with that of the normal human lens. The analyses were directed to ascertain, first, the dry weight and the total ash of the lenses examined, and, secondly, to determine quantitatively the percentage amounts of the potassium, sodium, calcium, magnesium, and silicon in the ash. The methods finally found most useful in the process of ashing and in the analyses for the different bases named are described below in some detail. It may be said in general that the calcium was precipitated as oxalate in acetic acid solution and this precipitate was afterwards dissolved in dilute sulphuric acid and titrated with potassium permanganate. The magnesium was precipitated as ammonia magnesium phosphate and weighed as magnesium pyrophosphate. The potassium was determined colorimetrically by means of the method of Cameron and Failyer as modified by Howell and Duke.[1] In the determinations of the sodium the greatest difficulty was experienced. The methods usually recommended in the literature were found unreliable. The method usually described is to remove, first, the calcium and magnesium and then precipitate the phosphoric acid left in the filtrate by means of barium hydroxide or silver oxide, the excess of barium or silver being removed subsequently as barium carbonate or silver chloride. Assuming that these separations were successful, the residue remaining should consist only of potassium, sodium, and ammonia salts. The last can be removed by incineration and the potassium and sodium be weighed as chlorides. Since the potassium had already been determined by the colorimetric method, it should then be possible to determine the sodium by difference. Unfortunately these methods, although carried out with the greatest care, gave variable results and often the ash remaining, which was supposed to

[1] Howell and Duke, *American Journal of Physiology*, vol. xxi., February 1, 1908.

consist of only the potassium and sodium chlorides, weighed more than the initial total ash of the lenses used for the determination. The difficulty lay apparently in the fact that when the phosphates were precipitated with barium chloride the excess of barium could not be removed without using at the end some sulphuric acid and in that case it was not possible to get the potassium and sodium wholly converted to chlorides. When silver oxide was used instead of barium hydroxide it was equally impossible to remove satisfactorily the last traces of the silver. After many fruitless efforts to get concordant results for the sodium determinations, the method described below was used with success. In this method the phosphates were precipitated by barium hydroxide, the excess of the barium was removed with sulphuric acid, and the potassium and sodium, after removal of the ammonia by incineration, were weighed finally as sulphates instead of chlorides. From this weighing the amount of sodium present could be determined since the amount of potassium was already known. Examination of the literature, so far as it has been accessible or known to me, shows that very little previous work of this kind has been done upon the crystalline lens. The variations in weight and size of the lens have been recorded by several observers (Smith,[1] et al.). They have brought out the interesting fact that the lens increases in weight and volume throughout life, but, according to Smith, there is no change in the specific gravity. Dor[2] states that the old lens contains less water than in youth, but that in cases of cataract the percentage of water is increased. Deutschmann[3] also states that in old age the percentage of water is diminished. Collins,[4] on the contrary, reports that in the old lens there is an increased percentage of water. This last observer studied also the variations in the ash of the lens, although he made no effort to analyze the ash into its several constituents. He finds that the ash of a healthy lens is nearly constant, amounting to about 1 *mg*.

[1] Smith, *Royal Ophthalmic Hospital Reports*, vol. x., 1880.
[2] Dor, *Rev. d'opht.*, April, 1907.
[3] Deutschmann, *Græfe's Arch. für Ophthalmologie*, xxv., 2, 1879.
[4] Collins, *Ophth. Rev.*, viii., S. 321–331, 1889.

Cataractous lenses contain relatively less water and more solids and ash than the normal lens. In the analyses made during the course of the present work the methods have varied from time to time. The final procedure adopted has proved very satisfactory, and since it differs in some respects from that used in similar investigations it may be well to give a brief outline.

PREPARATION OF MATERIAL FOR ANALYSIS.

The lenses are put into weighed platinum crucibles, these are placed in an air-bath, and the temperature of this is kept at 110° for 24 hours. At the end of this time the crucibles are put, while still hot, under a desiccator and finally weighed when cold. This weight minus the weight of the crucible is taken as the weight of the dry material.

The platinum crucibles with the dry material are transferred to porcelain crucibles and these are placed over Bunsen burners; the heat must be very gentle at first, and be gradually increased until the platinum crucibles come to a dull, red heat. This heating is continued until a white ash is obtained. The crucibles are removed from the flame and allowed to cool. A few drops of hot water are added to the ash, the crucibles are placed in an air-bath at 110° and when dry are transferred to the sand-bath and from this to the Bunsen burners. If there is then any appearance of carbon the heating is continued until a white ash is obtained. The platinum crucibles are again placed, while still hot, under a desiccator and finally weighed when cold. This weight minus the weight of the crucible is taken as the weight of the ash.

METHODS OF ANALYSIS.

To the ash are added five drops of a twenty-five per cent. solution of hydrochloric acid and this acid solution is evaporated to dryness over the water-bath. This process is repeated three times. The residue is dissolved in water and made up to 100*cc*. Of this quantity 10*cc* are taken for the determination of the potassium and the remaining 90*cc* for the determination of the calcium and magnesium.

(*A*). The 10*cc* are evaporated to dryness in a platinum crucible, five drops of a 25% solution of hydrochloric acid are added, and six drops of a 10% solution of platinum

chloride. This solution is evaporated just to dryness over a water-bath at 70°. This dried residue is washed five times, by decantation, with 95% alcohol. Each wash is poured into a centrifugalizing tube. The crucible with the part of the precipitate left in it is dried over the water-bath. The centrifugalizing tube with the remaining portion of the precipitate is placed in the centrifuge and centrifugalized. The supernatant liquid is siphoned off and about 5*cc* of alcohol are added to the precipitate and again centrifugalized. This process is repeated five times. The centrifugalizing tube is then placed in a beaker of hot water until the residue is dried. The precipitate in the centrifugalizing tube and crucible are dissolved in hot water and combined. To this solution a few drops of a 1% solution of hydrochloric acid are added. After the solution is cool, fifteen drops of a 25% solution of potassium iodide are added. A red color develops. After about fifteen hours this solution or a known portion is diluted until its color matches as nearly as possible that of a standard solution containing a known amount of potassium. Then in order to make a more accurate comparison of the two solutions Schreiner's colorimeter is used.

(*B*). The remaining 90*cc* of the dilute hydrochloric acid solution are evaporated on the water-bath to a small bulk. Ammonium acetate, ammonium chloride, and a small excess of ammonium oxalate are added. The precipitate, calcium oxalate, is thoroughly washed with hot water by decantation and centrifugalization, and is then treated with an excess of dilute sulphuric acid. This acid solution is heated to 60° and titrated with a dilute standard solution of potassium permanganate until the fluid just shows a red tint.

(*C*). The supernatant liquid and washings from the precipitate of calcium oxalate are evaporated on the water-bath to a small bulk, a slight excess of ammonium phosphate is added, and the solution is then made alkaline with ammonia and set aside for twenty-four hours. The magnesium is precipitated as magnesium ammonium phosphate. This precipitate is washed thoroughly with ammoniacal water by decantation and centrifugalization. It is then transferred to a platinum crucible; the latter, with the lid on, is exposed for some time to a very gentle heat, which is finally increased

TABLE I.

This table gives the details of the analyses of the ash of the normal human and pig's lens for potassium, calcium, magnesium, and sodium.

Pig	Age	Tissue	Dry Mat. in Mgms.	Ash in Mgms.	K. in Mgms.	Ca. in Mgms.	K. % in Dry Mat.	Ca. % in Dry Mat.	K. % in Ash	Ca. % in ash	Mg. in Mgms.	Mg. % in Dry Mat.	Mg. % in Ash.	Ash % of Dry Mat.
"	About 1	1 Lens	141.90	3.18	1.07		0.75		33.64					2.24
"	year	5 "	676.00	17.20							1.30	0.17	1.51	2.24
"	"	5 "	740.80	16.40							1.30	0.18	1.71	2.32
"	"	5 "	674.20	16.00							1.00	0.14	1.35	2.37
"	"	3 "	438.10	11.10							0.60	0.13	1.17	2.53
"	"	3 "	410.90	10.03							0.60	0.14	1.29	2.43
"	"	8 "	1187.30	28.90							1.70	0.14	1.27	2.43
"	"	7 "	992.60	23.60							1.20	0.12	1.10	2.37
"	"	5 "	659.90	17.00							0.80	0.12	1.01	2.57
"	"	5 "	663.40	16.20							0.80	0.12	1.06	2.44
"	"	1 "	132.00	3.10	1.00		0.75		32.25					2.34
"	"	1 "	134.00	3.00	1.00		0.74		33.33					2.23
"	"	1 "	136.00	3.20	1.10		0.73		34.37					2.35
"	"	1 "	140.80	3.20	1.22		0.86		38.12					2.27
"	"	1 "	137.60	3.30	1.09		0.79		33.03					2.39
"	"	1 "	139.70	3.30	1.26		0.90		38.18					2.36
"	"	6 "	791.60	19.00	6.40		0.80		33.68					2.40
"	"	6 "	816.90	19.80	6.47		0.79		32.70					2.42
"	"	58 "	7830.70	189.40		0.208		0.002		0.10	2.018	0.025	1.06	2.41
"	"	51 "	7259.50	179.10		0.167		0.002		0.09	1.909	0.020	1.05	2.46

Pig	Age	Tissue	Dry Mat. in Mgms.	Ash in Mgms.	K. in Mgms.	Ca. in Mgms.	K. % in Dry Mat.	Ca. % Dry Mat.	K. % in Ash.	Ca. % in Ash.	Mg. in Mgms.	Mg. % in Dry Mat.	Mg. % in Ash.	Ash % of Dry Mat.
"	"	22 lenses	3028.70	81.85		0.066		0.002		0.08	0.868	0.028	1.06	2.70
"	"	54 "	7367.65	189.95		0.133		0.001+		0.07	2.170	0.029	1.01	2.57
"	"	56 "	7748.30	179.05		0.133		0.001+		0.07	2.150	0.027	1.20	2.31
Human	67	normal "	81.00	2.05	0.827		1.02	40.36						2.53
"	67	" "	75.90	1.90	0.802		1.05	42.23						2.50
"	24	" "	50.00	1.10	0.545		1.09	49.54						2.20
"	24	" "	50.00	1.10	0.527		1.05	47.90						2.20
"	65	Glaucoma. "	69.05	1.90	0.617		0.89	32.50						2.75
"	25	" "	45.75	1.00	0.301		0.65	30.08						2.18
"	35	" "	54.80	0.85	0.294		0.53	34.58						1.55
"	35	" "	55.80	1.05	0.294		0.52	28.00						1.88
"	42	" "	64.65	1.80	0.731		1.13	40.61						2.78
"	42	" "	43.00	0.85	0.366		0.85	43.05						1.97
"	0	2 " "	33.10	0.85	0.222		0.67	26.09						2.56
"	0	2 " "	34.25	0.45	0.127		0.37	28.22						1.21
"	0	2 " "	31.30	0.40	0.159		1.20	39.75						1.27
"	0	2 " "	25.15	0.35	0.103		0.40	29.42						1.39

TABLE II.

This table gives the details of the analyses of the ash of the cataractous lens (United States) for the potassium, calcium, and magnesium.

Human	Age	Tissue	DryMat. in Mgms.	Ash in Mgms.	K. in Mgms.	Ca. in Mgms.	K. % in Dry Mat.	Ca. % in Dry Mat.	K. % in Ash	Ca. % in Ash	Mg. in Mgms.	Mg. % in Dry Mat.	Mg. % in Ash	Ash % of Dry Mat.
"	70	cataract. lens	31.90	0.80		0.088		0.27		11.00				
"	70	" "	59.05	1.00		0.143		0.24		14.30				
"	64	" "	35.60	0.80	0.170		0.47		21.25					
"	70	45 " "	1131.10	28.80		3.250				11.28	1.340	0.11	4.65	2.54
"	70	44 " "	1173.95	22.25		2.940				13.21	1.324	0.11	5.94	1.89
"	70	10 " "	445.60	5.20		0.881				16.94	0.347	0.08	6.67	1.16
"	70	10 " "	533.90	5.40		0.971				17.89	0.520	0.10	9.62	1.01
"	70	10 " "	423.30	4.70		0.813				17.29	0.477	0.11	10.14	1.11
"	10	11 " "	401.20	5.60		1.050				18.85	0.282	0.06	5.03	1.21
"	70	00 " "	433.80	3.80		0.621				16.34	0.282	0.06	7.41	0.87
"	70	00 " "	370.20	3.90		0.695				17.81	0.347	0.08	8.89	1.05
"	70	71 " "	377.20	3.80		0.599				15.76	0.521	0.13	13.71	1.00

Human	Age	Tissue	Dry Mat. in Mgms.	Ash in Mgms.	K. in Mgms.	Ca. in Mgms.	K. % in Dry Mat.	Ca. % in Dry Mat.	K. % in Ash	Ca. % in Ash	Mg. in Mgms.	Mg. % in Dry Mat.	Mg. % in Ash	Ash % of Dry Mat.
"	67	cataract. lens	42.00	1.00	0.110	0.090	0.26	0.21	11.00	9.00				2.37
"	75	I " "	44.20	1.20	0.110	0.097	0.24	0.21	9.16	8.08				2.71
"	64	nucleus " "	22.00	0.50	0.090	0.045	0.40	0.20	18.00	9.00				2.27
"	64	cataract. "	51.65	1.00	0.162	0.106	0.31	0.19	16.25	10.60				1.93
"	78	I " "	32.00	1.00	0.110	0.100	0.34	0.31	11.00	10.00				3.12
"	70	I " "	34.20	1.12	0.102	0.100	0.29	0.29	9.10	8.92				3.27
"	70	I " "	38.95	0.90	0.070	0.110	0.18	0.28	7.80	12.22				2.31
"	70	16 " "	557.40	21.40	1.400	2.940	0.25	0.52	6.50	13.73				3.83
"	70	9 " "	252.80	6.25	0.720	0.531	0.28	0.21	11.52	8.50				2.47
"	70	I " "	23.80	1.20	0.080	0.137	0.32	0.57	6.66	11.49				5.04
"	70	I " "	83.15	2.80	0.300	0.180	0.36	0.21	10.71	6.42				3.36
"	80	I " "	24.10	0.60	0.038	0.06	0.15	0.24	6.45	10.00				2.48
"	75	I " "	30.85	0.90	0.028		0.09		3.16					2.91
"	70	I " "	34.70	0.85	0.116		0.33		13.67					2.44
"	70	I " "	50.87	1.10		0.143		0.28		13.00				2.16
"	70	I " "	53.80	1.65	0.057		0.10		3.45					3.06
"	70	I " "	23.65	0.70	0.039		0.16		5.70					2.96
"	70	I " "	52.75	1.00	0.051		0.97		5.13					1.89

TABLE III.

This table gives the details of the analyses of the portion of the ash, soluble in dilute hydrochloric acid, of the cataractous lens (India) for the potassium, calcium, magnesium, and sodium.

Species	Age	Tissue	Dry Mat. in Mgms.	Ash in Mgms.	K. in Mgms.	Ca. in Mgms.	Mg. in Mgms.	Na_2SO_4 and K_2SO_4 in Mgms.	Na. in Mgms.	K. % in Dry Mat.	K. % in Ash	Na. % in Dry Mat.	Na. % in Ash	Ca. % in Ash	Ash % in Dry Mat.	Mg. % in Ash
Human		cataract. lenses from India	2288.25	33.90	2.04	2.17		32.00	8.14	0.09	6.01	0.35	24.01	6.40	1.48	
"		" "	2288.25	33.90	2.00	1.96		32.20	7.88	0.09	5.81	0.34	23.27	5.80	1.48	
"		" "	9283.40	147.10	10.86	7.35		152.30	41.05	0.11	7.38	0.44	27.90	5.00	1.58	
"		" "	3206.50	53.50	2.60	3.58	0.97				4.85			6.69	1.6	1.81
"		" "	3564.60	73.40	3.89	4.18	1.05				5.29			5.69	2.0	1.43
"		" "	4310.70	70.90	4.00	5.04	1.18				5.64			7.10	1.6	1.66
"		" "	4374.20	90.20	3.96	5.08	1.22				4.39			5.63	2.0	1.35
"		" "	4782.10	81.00	4.82	5.53	1.40				5.95			6.82	1.6	1.72

TABLE IV.

This table gives the details of the analyses of the portion of the ash, insoluble in concentrated hydrochloric acid, of the cataractous lens (India) for calcium, potassium, magnesium, and silicon.

Dry mat. in Mgms.	Ash in Mgms.	Wt. of mat. insol. in con. HCl. in the ash in Mgms.	Wt. of Si. in the insol. mat. in Mgms.	Wt. of Ca. in the insol. mat. in Mgms.	Wt. of K. in the insol. mat. in Mgms.	K. % in insol. mat. of dry mat.	K. % in the insol. mat. of the ash	Ca. % in insol. mat. of dry mat.	Ca. % in insol. mat. of ash	Si. % in insol. mat.	Percent. of ash sol. in dilute HCl.	Percent. of ash insol. in concentrated HCl.	Percent. of Si. in ash
3206.50	53.50	8.60	1.54	0.17	0.82	0.02	1.53	0.005	0.31	17.90	83.92	16.08	2.87
3564.60	73.40	27.00	3.91	0.60	2.86	0.08	3.89	0.01	0.81	14.48	63.21	36.79	5.32
4310.70	70.90	12.90	2.26	0.23	1.20	0.02	1.69	0.005	0.32	17.51	81.80	18.20	3.18
4374.20	90.20	35.60	5.13	0.82	3.42	0.07	3.79	0.01	0.90	14.41	60.53	39.47	5.67
4782.10	81.00	18.20	2.70	0.35	1.90	0.04	2.33	0.007	0.43	14.83	77.53	22.47	3.33
5367.70	90.90	17.00	2.64	0.36	1.81	0.03	1.93	0.006	0.39	15.52	81.29	18.71	2.90
4679.10	77.20	11.40	1.67	0.23	1.18	0.02	1.52	0.004	0.29	14.64	85.23	14.77	2.16

to intense redness. The crucible is put, while still hot, under a desiccator, and finally when cold the magnesium is weighed as magnesium pyrophosphate.

(*D*). The supernatant liquid and washings from the precipitate of ammonium magnesium phosphate are evaporated on the water-bath to a convenient bulk, a slight excess of baryta water is added, and the solution is boiled. This precipitate is thoroughly washed with warm water by decantation and centrifugalization. The original liquid and washings are evaporated to a few *cc* and the barium removed with sulphuric acid. The precipitate, barium sulphate, is thoroughly washed by decantation and centrifugalization. The supernatant liquid and washings are evaporated to a small bulk poured into a platinum crucible, and evaporated to dryness over a water-bath. The ammonium salts are removed by ignition at a low red heat. A few drops of a 5% solution of sulphuric acid are added to the residue, which is then evaporated to dryness and heated to a very dull red heat to drive off all free sulphuric acid. The crucibles are put, while still hot, under a desiccator and the alkalies, sodium and potassium, are weighed as sulphates. The potassium in this residue was determined by the method previously described, and the sodium was obtained by the difference. The determinations of the potassium made in this residue agreed very well with those made upon an aliquot part of the solution of the original ash (*A*).

RESULTS.

The main facts brought out by these analyses may be presented briefly in the following table, which gives the average results as estimated for a single lens.

	Average dry wt. of one lens	Average wt. of ash of one lens	Percent. wt. of ash to dry wt.	Percent. of K. in ash	Percent. of Ca. in ash	Percent. of Mg. in ash	Percent. of Na. in ash	Percent. of Si. in ash
Normal adult human lens.	58.99 mgr.	1.40 mgr.	2.30	38.80	?	?	?	0
Normal adult pig's lens	137.70 "	3.40 "	2.45	34.30	0.08	1.20	6.67	0
Embryo human lens	15.47 "	0.25 "	1.60	30.80	?	?	?	0
Cataract human lens (United States)	34.42 "	0.58 "	1.68	9.80	12.50	8.00	23.82	0
Cataract human lens (India).	92.30 "	1.52 "	1.64	5.81	6.00	1.6	25.06	3.63

On the basis of these figures the following general conclusions may be drawn.

1. In the human cataractous lens (senile cataract), the percentage of potassium in the ash is greatly reduced, while the percentages of calcuim and sodium are greatly increased, as compared with the normal lens. In the lenses obtained in the United States, there was also a distinct increase in the magnesium in the ash, while in those from India there was a notable amount of silicon. It will be noted that no figures were obtained for the calcium, magnesium, or sodium for the normal human lens, owing to the difficulty in getting this latter material. It is certain, however, that in the normal human lens the calcium and the magnesium exist in very small amounts, as proved by quantitative reactions, and although the amounts were too small for quantitative determination it seems most probable that they are of the same order as in the normal pig's lens. Sodium determinations were not made for the normal human lens, since at the time that this material was being analyzed a satisfactory method of determining sodium had not been obtained. For this element also it is necessary in the comparison with the cataractous lens to use the figures obtained from the pig's lens. In regard to the cataractous lenses obtained from India (Dr. Smith), we have the additional peculiarity of the presence of a distinct amount of silicate of calcium and potassium and possibly sodium. In the cataractous lenses obtained from the United States (Dr. Greene), silicates did not occur, or at least did not occur in quantities sufficient for determination. The ash of a large number of these lenses was either completely soluble in dilute hydrochloric acid, or if any residue remained it was so minute as to be beyond the possibility of analysis. What conditions cause such a marked deposition of silicates in the cataractous lens of the inhabitants of India can not be stated. The natural suggestion is that there may be some peculiarity in the diet to account for the silicate, and it would be interesting to ascertain whether other organs or the liquids of the body in these individuals also contain notable amounts of silicates.

2. It is worthy of special comment that the changes in the ash of the cataractous lens are of such a character as to

bring it more closely in connection with the composition of the blood and lymph. That is to say, in cataract, the lens loses the characteristic ash content of a living tissue and approaches the composition of the liquids of the body to the extent that its content in potassium is diminished and its content in sodium and calcium is increased.

3. The figures for the cataractous lens indicate that as compared with the normal human lens it contains less organic material and less percentage of ash. Unfortunately, no figures were obtainable in regard to the average total weight and water contents of such lenses, but if Collins is correct in stating that the cataractous lens has in general a higher percentage of solids and ash in reference to the total weight, the figures obtained in my analyses can only indicate that the total weight of the cataractous lens must be smaller than that of the normal lens.

4. As between the normal human lens and the normal pig's lens it is interesting to note that although the pig's lens is much larger and heavier and contains much more solid material and ash, nevertheless the percentage of ash is identical in the two cases, and so far as the analyses were carried the composition of the ash is apparently the same.

5. It was hoped in the beginning of the work that sufficient normal human material might be obtained to furnish data in regard to the changes in ash content, if any, with increasing age. As a matter of fact, a quantitative estimation of the ash in the normal human lens was possible only for the potassium, and so far as this element is concerned the analyses indicate that there is no change with age. The lens in old age as compared with the lens in the embryo shows certainly no diminution in potassium, and most probably there is no increase in calcium or magnesium, since in the old as in the young lens these elements are present in such small quantities as to make a quantitative determination impossible, unless a very large number of lenses are used. On the basis of these facts, one may conclude that the lens in the very old (70 years) shows no such evidence of senility, when judged by its mineral constituents, as is seen in some other tissues, the arteries, for example, and this conclusion falls in with the known fact that the lens, unlike most of the organs, continues to grow through-

out life. This fact would also indicate that it is a mistake to assume that senile cataract is an instance of premature senility in the lens, that is to say, that it is simply an acceleration of a normal senile change. On the contrary, the evidence, so far as it goes, would indicate that senile cataract when it occurs is due to some cause which has interrupted the normal metabolism and brought on a moribund condition of the lens tissue.

6. It will be noted that in the lens of the embryo the total solid material is small and the percentage of ash is also small. The growth of the lens that is known to take place throughout life is indicated by the few figures in the table which give the weight of solid material at different ages.

7. In Table IV. it will be noticed that 76.21 per cent. of the ash of the lenses obtained from India was soluble in dilute hydrochloric acid, and that 23.79 per cent. was insoluble in concentrated hydrochloric acid. This insoluble portion of the ash was analyzed for magnesium, silicon, calcium, and potassium. No magnesium was found. Silicon was present to the extent of 15.61% of the insoluble material, calcium to 0.49 % of the total ash, and potassium 2.38% of the total ash. Hence, in these lenses there are present silicates of calcium and potassium and possibly of sodium.

Tables II. and III. show that the percentage of calcium and potassium in the ash of lenses obtained in the United States is greater than that in the ash, soluble in dilute hydrochloric acid, of the lenses obtained from India. However, if the amount of calcium and potassium found in the silicate of the ash of the Indian lenses be combined with the calcium and potassium content of the part of the ash soluble in dilute hydrochloric acid, then the percentage of calcium and potassium in these lenses is found to be about the same as that of the lenses from the United States.

SUMMARY.

1. There is a decrease of potassium in the cataractous lens from 38.8% of the ash in the normal to 9.8% in cataract.

2. The calcium in the cataractous lens increases from an almost negligible quantity in the normal to 12.5% in cataract.

3. There is an increase in the magnesium in the cataractous lens obtained in the United States from 1.20% as estimated in the pig's lens to 8.00%. This increase is therefore not so marked as the increase in the calcium.

4. Assuming that the sodium in the normal pig lens is about the same in quantity as the sodium in the normal human lens, then the increase in sodium is practically of the same extent as the decrease in the potassium, the sodium increasing from 6.67% in the normal to 25.06% in the cataractous condition.

5. The lenses obtained from India differ from those obtained from the United States in that they contain a large amount of calcium potassium and possibly sodium silicate, and fail to show any increase in the percentage of magnesium in the ash.

At this place I desire to thank Professor W. H. Howell, at whose suggestion this work was begun and under whose direction it has been carried on, for his kindly criticism and interest.

ALTERATIONS IN THE COLOR FIELDS IN CASES OF BRAIN TUMOR.[1]

By Dr. JAMES BORDLEY, Jr., and Dr. HARVEY CUSHING,
Baltimore.

(With twenty-one charts on eight figures in the text.)

ACCORDING to Parinaud hysterical amblyopia has been recognized since early in the 17th century, when it was clearly described by Charles Sapois. It was regarded by von Graefe, in 1865, as a true anæsthesia of the retina, and a few years later Charcot and Labor pronounced the so-called functional defects of vision cerebral, rather than retinal lesions. Dyschromatopsia—a condition especially investigated by Galezowski, who with Charcot was most instrumental in bringing about the wide recognition of these states—was regarded by them, and later by Parinaud, as a manifestation of hysteria, especially when associated with concentric contraction of the visual fields. This is an impression, we think, which still holds sway to-day.[2]

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[1] Presented by Dr. Cushing at a meeting of the American Neurological Association, New York, May 27, 1909.

[2] One of our recent patients in whom a temporal lobe tumor was disclosed at operation has in her possession a letter from a distinguished neurologist, written only a few days before the operation. From it we take the liberty of quoting a few sentences:

" . . . I had the eyes examined in the first place in the fear of there being some choroidal changes due to nephritis, and incidentally the fields of color and vision were taken. A little to my astonishment we found complete reversal of the red, green, and blue fields with great contraction. This is a condition which I think may be said to be thoroughly characteristic of hysterical trouble and to be exceptionally rare—*almost unknown—in any other condition* [italics ours]. Additional suggestions of this same condition are these: the corneal re-

We find, upon a careful review of the extensive literature, that every conceivable alteration in the form and color fields has been ascribed to hysteria; but there are two conditions emphasized as being distinctly pathognomonic; one of them the view of the Charcot school, which attributes to these states the concentric contraction of the field for form with partial or complete color reversal (dyschromatopsia); and, the other, the view that hysterical subjects have a predilection for red—merely another way of saying that the red field may encircle the blue or the blue become inverted.

Granted that these changes are actually characteristic of hysteria, it appears that they are no less so for brain tumors; and whether a similar condition exists as the intracranial basis for the symptom in each, can only be conjectured. We at least must face the disconcerting fact that almost every patient brought for operation, after the diagnosis of brain tumor has finally come to be read by the ophthalmoscope on his retina, has at one time or another been regarded as hysterical or the subject of some psychoneurosis. And it must be realized that most brain tumors run a course of years rather than months—this contrary to the prevailing opinion. A case may be cited in illustration:

> A young woman, twenty-nine years of age, the wife of a physician, had suffered for years from periodic attacks associated with states of hemianæsthesia, peculiar sub-

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flexes are subnormal and the whole left side has subnormal sensation to pain, etc.

". . . While these hysterical symptoms are very distinct, and, as I said, due in my experience to no other condition, it seems to me at least probable that there is also a genuine slight loss of power on the left side, probably from a stoppage in a superficial vessel; and to this there have been added these hysterical manifestations. They were rather unexpected to me, as Mrs. —— seems to be a person with self-control and calm decision. . . . I should treat her with massage . . . to prevent any tendency to contracture, which follows as certainly on hysterical paralyses as on cerebral or spinal ones."

This letter we think is fairly representative of the views which are generally held in regard to these matters.

Doubtless disturbances of the color fields have been observed heretofore in patients known to have brain tumor, but so far as we are aware they have never been commented upon as being characteristic of any condition other than hysteria.

jective sensations of taste and smell and occasionally by convulsive seizures. Owing to a tendency to amenorrhœa the symptoms had been attributed to some pelvic disorder until operative explorations had shown this conjecture to be incorrect.

The seizures were said to be worse in the spring and fall than at other seasons; at least they had been definitely fluctuating during the ten years of their occurrence. Being a physician's wife she had been seen by many specialists, each consultation leading almost invariably to a diagnosis of hysteria. The patient herself said that she had little doubt as to the correctness of this view for her sister was also "hysterical."

A sudden, rather sharp augmentation in all of these long-standing symptoms was associated with profound headache, vomiting and the onset of choked disk with a complete homonymous hemianopsia and color inversion. A simple decompression was performed, with prompt relief to all pressure symptoms, and later on the occipital lobe was thoroughly explored in the hope that a lesion might be disclosed. The symptoms were alleviated for a year's time, when after a careful review of her history it became apparent that the whole picture was a definite one of temporal lobe lesion with involvement of the uncinate gyrus. The region was exposed and a large tumor which had almost completely degenerated was disclosed.

When we began our conjoint observations of cases of brain tumor in 1902, among our early cases we met with an occasional instance of color reversal which we were naturally inclined to attribute to some associated functional element; but as their number increased, with the frequent demonstration of an actual organic lesion, and as most of the patients with these color changes showed no other clinical indication of functional disturbance, we gradually came to look upon dyschromatopsia as a symptom often of great value, particularly in an early stage of brain tumor. A few characteristic fields have been selected from our several hundred charts taken during the past seven years, which will serve to illustrate the main points we wish to emphasize.

We have found no definite relationship between the contraction of the field for form and that for colors. Some of the patients have shown a concentric contraction with color reversal (Fig. 1), such as Parinaud regards as the "essential

sign of hysteria," but the usual early return of the normal relations after the pressure symptoms have been relieved by operation suffices to controvert the view of their being hysterical, in the usual application of this term.

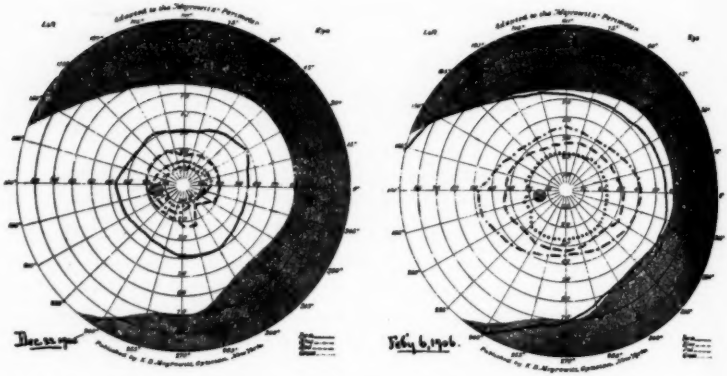

FIG. 1 (Surg. No. 20996). Illustrating concentric contraction with color reversal, a common form of disturbance. Conditions were similar in the right eye.

Patient with typical tumor symptom-complex, choked disk, etc. Myopia; vision corrected, R = $^{20}/_{30}$; L = $^{20}/_{50}$. Fields were taken (Chart 1) December 22, 1905. Operation December, 25 1905. February 6, 1906, fields normal (Chart 2), no swelling of nerve heads; vision corrected, R = $^{20}/_{20}$; L = $^{20}/_{20}$.

The patient from whom these charts (Fig. 1) were made was distinctly not an hysterical subject, though the perimetric fields were so suggestive that she was kept under observation by her physician for nearly two years before an operation was performed. Recovery in her case was prompt, and she is to-day earning her livelihood as a seamstress; her vision and fields show not the slightest defect.

In another group of cases the patients have shown not only concentric contraction of the form fields in association with color reversal, but likewise peripheral limitations which are relatively greater in the form than in the color fields, and in these cases red is apt to be less affected than blue (Fig. 2). Hansel and Sweet have been credited by Parker with the statement that this is "the predominant peculiarity of an hysterical contraction of the visual fields; . . . it cannot be simulated and is caused by no other disorder."

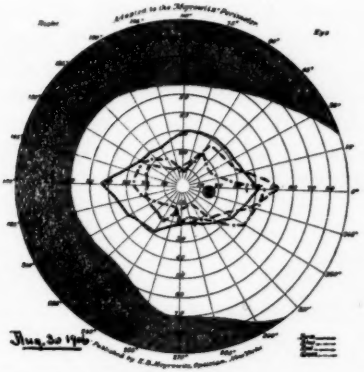

FIG. 2 (Surg. No. 15893). Showing characteristic contraction, greater in the form than in the color field, in addition to color and form interlacing.

The patient, a young girl having focal palsy, in addition to the characteristic pressure phenomena of tumor with a choked disk of 3 D., etc. Complete restoration of normal relations followed a simple decompression which disclosed a brain under tension.

There have been other cases in which the contraction for colors and form goes hand in hand, with the exception that the blue is almost invariably much more affected than the red field (Fig. 3). Indeed, the prominent feature in most of our

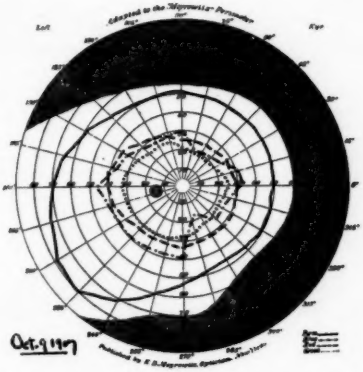

FIG. 3 (Surg. No. 21400). To illustrate fairly equal shrinkage of form and color fields. Note tendency of the blue field to become inverted through interlacing. Patient with a right cerebellar cyst producing internal hydrocephalus. Disks swollen 4½ D. before operation when chart was plotted. Choked disk has virtually cleared up thirteen days after right subtemporal decompression.

charts is not so much the preservation of the red field as the alteration in the blue. We have seen the green circle outside of the red and the red outside of the blue, but there is invariably an alteration in the position of the blue which exceeds that of the other colors. In fact blue blindness has been the sequel in several cases in which the color change has been rapidly progressive.

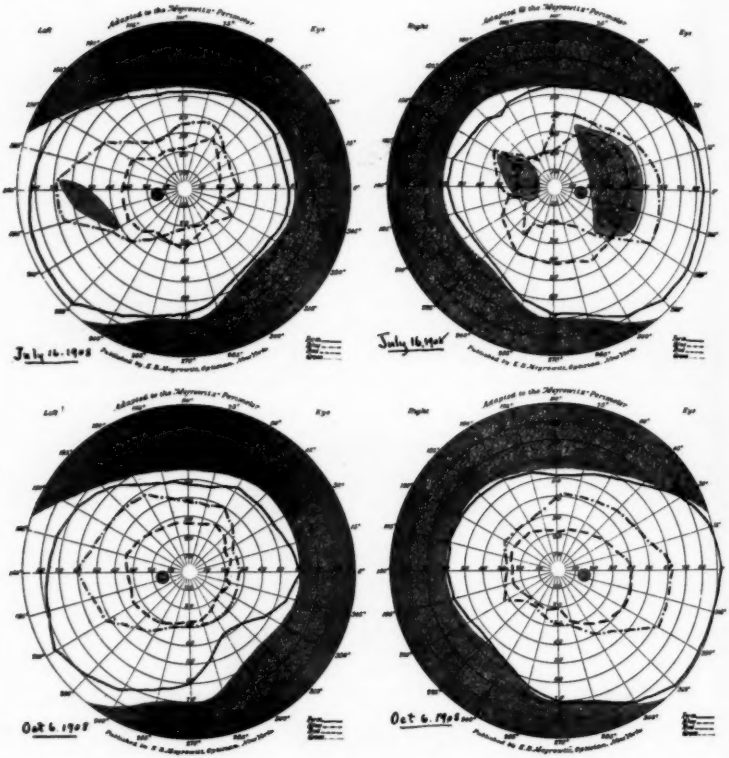

FIG. 4 (Surg. No. 23018). To illustrate possible manner of formation of constrictions of the blue field, by the occurrence of peripheral scotomata for blue (shade areas); green field not charted.

A case of left cerebello-pontine tumor. First series taken July 16, 1908; choked disk 3 D. Operation July 17th, with rapid restoration of normal conditions, disappearance of scotomata, and return of color outlines to usual relations. Compare series of Oct. 6, 1908.

In certain cases we have had the good fortune to observe the manner of constriction of the blue field, the contraction apparently being brought about by the formation of peripheral scotomata which gradually became confused (Fig. 4). Whether this is the usual manner in which blue is lost we can of course only conjecture. We have never observed scotomata for colors other than blue; it may be added that we have limited our tests to blue, red, and green.

These findings merely serve to illustrate that the changes of most significance, in tumor at all events, are possibly to be looked for in the field for blue. This may serve to be of certain differential value, since tabes, multiple sclerosis, etc., show a primary change which is often limited to the red field. Indeed it has been said that a disturbance in the red field is an indication of organic disease; changes in the blue field of functional disturbance.

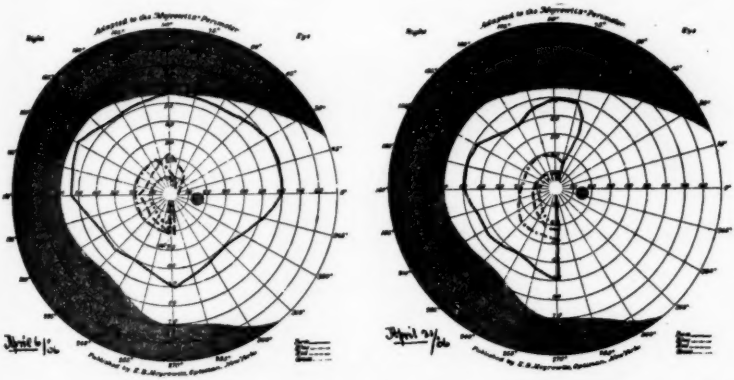

FIG. 5. Illustrating the progress of hemianopsia in the right eye alone, the condition having been homonymous for the two eyes.

Chart 1, taken shortly after a simple decompression operation showing not only the hemiachromatopsia, but also definite color reversal.

Chart 2, showing the condition two weeks later. Note the complete hemianopsia which has occurred by this time; also, in company with the disappearance of general pressure symptoms, the restoration of the normal relations of the color fields, which, however, remained constricted.

Charts were taken of this patient about once a week for several months, showing the gradual changes which led up to these two figures.

On May 24th focalizing symptoms have become evident through the right asteriognosis, etc. An exploration revealed the presence of a large glioma in the left parietal region which evidently compromised the left optic radiation.

Complete achromatopsia has occurred in a number of instances. (*Cf.* Fig. 8.) Patients with this condition have suffered from the so-called "India ink sketch" vision, which leads to greater visual confusion than is associated with

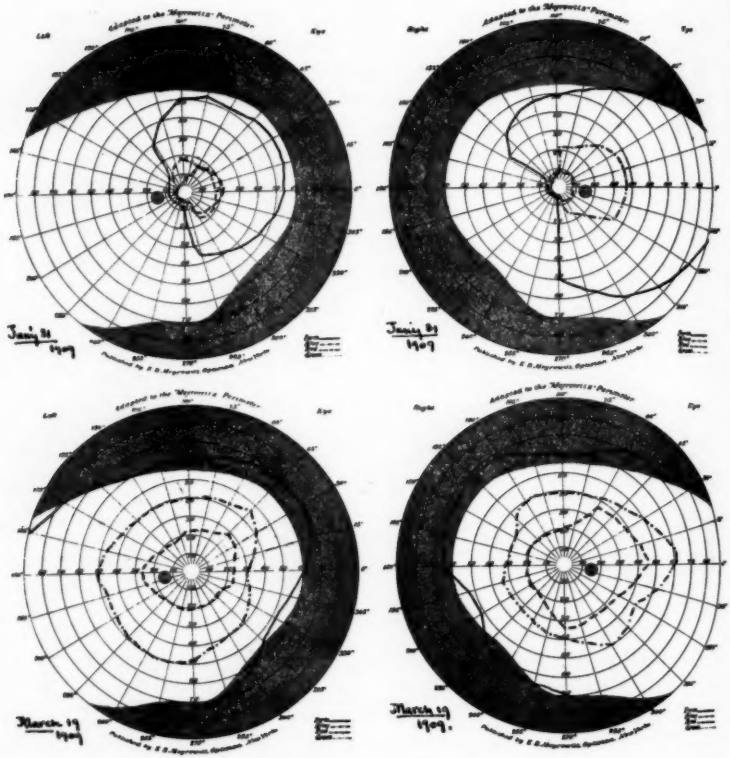

FIG. 6 (Surg. No. 23551). Charts taken before and after operation: patient with a left temporal lobe cyst, which was evacuated with complete disappearance of all symptoms.

The first series of charts, taken January 31, 1909, show a more complete homonymous half blindness for color than for form. Note on the left tendency to interlacing of the blue field. Vision, L = $^{20}/_{30}$; R = $^{20}/_{60}$. Choked disk, right, 3 D.; left, 4 D. Operation February 2, 1909.

Second series of charts, February 12th, showed total disappearance of hemianopsia and tendency of color fields to assume normal configuration. Practical subsidence of choked disk. Vision, L = $^{20}/_{30}$; R = $^{20}/_{20}$.

Third series of charts, March 19th, practically normal condition. Absence of choked disk. Vision, R = $^{20}/_{15}$; L = $^{20}/_{15}$.

even marked constriction of the field for form. Their vision is much more reduced than the ophthalmoscopic picture and the degree of constriction of the form field would lead one to expect.

The changes in the color fields, furthermore, often foretell the alterations which subsequently are to take place in those for form. Thus hemiachromatopsia has occasionally been followed by complete homonymous hemianopsia involving the same side. In one instance we have been able to follow the progress of these changes which occurred in the course of a few weeks. (Fig. 5.) Soon after the hemiachromatopsia was established in this patient the visual acuity of the corresponding area gradually decreased as the form outline slowly moved in toward the mid-line. The centre of fixation was bisected by the line of separation in this case only; in the other cases of hemiachromatopsia which we have seen the color lines have cleared the centre and encroached on the side that showed the color-blindness. (Fig. 6.)

It should be emphasized that the color disturbances do not

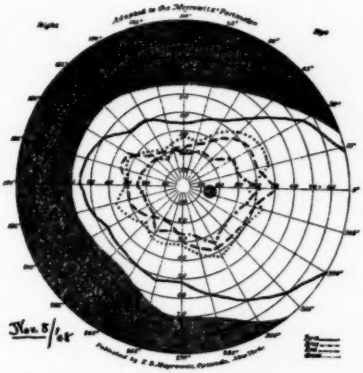

FIG. 7. (Surg. No. 23150). To illustrate the fields taken before operation for removal of a syphiloma of the right post-central region.

Patient had focal epilepsy with the usual evidence of general increase in pressure except for the absence of choked disk. Fields taken November 8, 1908. Operation November 11th; fields taken November 29th show normal color relations with restoration of normal confines both for form and colors.

seem to be dependent upon the presence of neuroretinal œdema, because in four of our patients they preceded any appreciable ophthalmoscopic changes (Fig. 7); and it may

be noted that we regard the early hyperæmia and congestion as a definite stage in the process of choked disk.[1] On the other hand, we must acknowledge that in six instances in our series of tumor cases the tumor symptom-complex has been unaccompanied by any alteration whatsoever in the visual fields.

While we have had the opportunity of examining about two hundred and fifty patients with brain tumor, we have chosen, for several reasons, to confine the statistics relating to our observations to fifty-six cases in the series. For in this particular group we have made a special effort to eliminate any possible source of error on the part of patient or observer. In doubtful cases our findings have been corroborated by others who were unaware of the fact that we were looking for color reversal; and there is abundant proof that inattention, poor light, diminution of the light sense and peculiarities in our color disks bear no relation to the results which we have obtained.

There have naturally been many patients in whom an examination of the visual fields was inconclusive or totally precluded, owing to blindness or mental or physical infirmity, and the observations are naturally impossible in the aphasic or in young children. Some of the examinations also were but roughly made, or the results rendered doubtful by the circumstances under which the perimeter was used. One particular source of possible error—faulty color disks—which was subsequently remedied, has led us to eliminate from the quoted series some of the earlier cases.[2]

The data derived from these fifty-six cases, owing to these special precautions, may therefore be of especial value. The figures relate to the conditions which were present at the time of the first examination, often before the diagnosis of tumor was certified:

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[1] Bordley and Cushing: "Observations on Choked Disk, etc." *The Journ. of the Amer. Med. Assn.*, 1909, lii., p. 353.

[2] After a careful examination of the ordinary disks sold with perimeters, comparing them with standardized and saturated colors, we found that disks supposed to be of the same color were often radically different. Indeed disks with pure colors were rarely found. A green tinge in the blue disk from admixture of yellow is a particular source of error. We are indebted to Dr. N. M. Black of Milwaukee for some specimens of pure spectral colors.

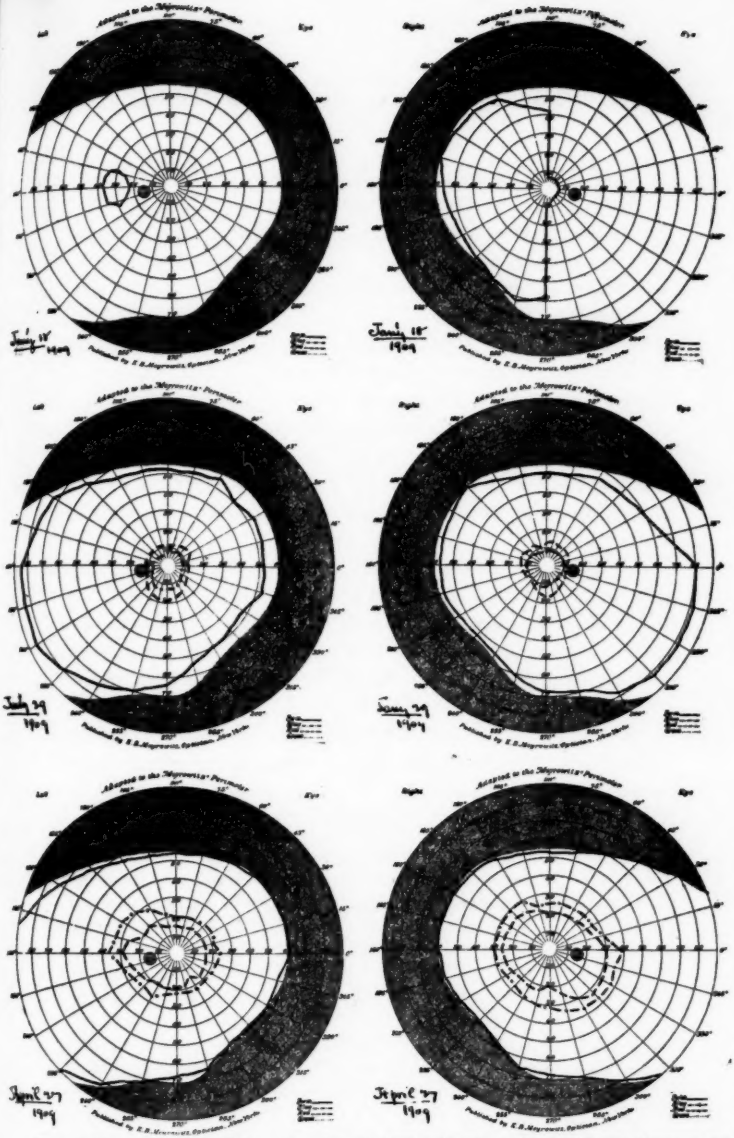

FIG. 8 (Surg. No. 23482). A series of charts showing the results of three examinations in a patient subjected to a simple left subtemporal decompression for a presumed tuberculoma of the left occipital lobe. Headache, vomiting, and moderate grade of choked disk (1½ D.) were present before the first operation.

January 18, 1909. Charts showing complete achromatopsia hemianopsia in the right and a very small area of sight remaining in the left. Vision, R = $^{8}/_{200}$; L = fingers at a few inches.

Second series of charts, January 29th, showing return of normal outlines for form; color perception limited to small central vision with almost complete inversion for blue. Vision, R = $^{12}/_{200}$; L = $^{8}/_{200}$. Subsidence of all pressure symptoms at this time.

Subsequent charts, by Dr. N. M. Black of Milwaukee, taken March 17, 1909, and April 27th, show gradual return to normal relations and widening of color fields. Vision $^{20}/_{30}$ in both eyes. All evidences of pressure have disappeared, and the rapid loss of the hemianopsia is presumably accounted for by the lifting away from the optic radiation of the presumed lesion which remains in a quiescent state.

	CASES
1. Interlacing of color lines the predominant feature	25
2. Color inversion the predominant feature	9
3. Hemiachromatopsia without corresponding change in form field	4
4. Islands of blue blindness (scotomata)	3
5. Blue blindness	3
6. Complete green blindness	1
7. Complete achromatopsia	5
8. No color change whatsoever	6

The striking features are: (1) The fact that in many of the charts the fields for form are unchanged, while there is a very definite change in the fields for color. Fifty out of the fifty-six cases show color changes; only eighteen show definite form changes.

(2) In four of the cases in which the colors interlaced tumors were found, though there was no choked disk.

(3) In forty-one out of forty-two cases examined subsequent to operation, whether palliative or radical, the color lines became restored to their normal relative position. (For a striking illustration see Fig. 8.)

The dyschromatopsia seems to depend in some fashion upon an increase of intracranial tension, the relief of which usually causes its early subsidence, and it is possible therefore that it may characterize organic lesions other than tumor.[1]

[1] Since these statistics were compiled we have had additional evidence, from sources other than our own clinic, of the correctness of the view that these changes in the color fields, so long looked upon as peculiar to hysteria, occur with great regularity in patients suffering from the tumor symptom-complex.

CONCERNING A FORM OF RETINAL DISEASE, WITH EXTENSIVE EXUDATION; BEING A CLINICAL CONTRIBUTION, WITH THE REPORT OF TWO CASES.[1]

By Dr. G. E. DE SCHWEINITZ, Philadelphia.

With two text figures and a drawing on Text-Plate XXVIII.)

THE following case histories are intended as a brief clinical contribution to a class of cases in which the lesions in the fundus of the eye are chiefly characterized by an extensive mass of exudation, sometimes associated with vascular disease. The whole subject has recently formed the basis of an admirable paper by George Coats,[2] who has gathered together the scattered literature and recorded pathological examinations of six cases that have come under his notice.

Case 1.—H. H., female, aged nineteen, unmarried, came for examination October 19, 1907.

Family History.—The patient's paternal grandfather died of softening of the brain after an illness of ten days, at the age of forty-four. The paternal grandmother died of diabetes mellitus, after an illness of four months, at the age of seventy-six. The maternal grandfather died of cardiac failure at the age of eighty, and the maternal grandmother of apoplexy at the age of sixty-seven. The patient's father and mother are living and in good health. The mother has had one miscarriage. One of the patient's uncles died of pulmonary tuberculosis at the age of fifty-two, another of cirrhosis of the liver at the age of forty; one aunt and three uncles are living and in good health. Several grand-uncles and aunts died of apoplexy or have suffered

[1] Read before the American Ophthalmological Society, July 14–15, 1909.

[2] *Royal London Ophthalmic Hospital Reports*, xvii., part 3, 1908.

from paralysis. There is no history of syphilis or alcoholism in the family.

Personal History.—The patient in childhood had diphtheria, measles, chicken-pox, and whooping-cough, and a number of attacks of malaria, but none of these occurred during the past five or six years. Menstruation began at the age of thirteen and has been quite regular, except at the age of sixteen, when for a short period of time there was a certain amount of irregularity. There have been several attacks simulating appendicitis.

General Examination.—This was made by Dr. David Riesman, who has kindly furnished the following notes: The patient is pale and has a slightly greenish tint. The heart's action is rapid, but there is no murmur; pulse 120; temperature 98.6°; examination otherwise negative. The hemoglobin 76 by Dare, 80 by Talquist; red cells 4,365,000, white cells 8,400. Differential count: Polymorphonuclears 66, lymphocytes 27, large mononuclears 6.4, eosinophiles 0.2; one nucleated red cell. Otherwise nothing abnormal.

Urine Examination.—Color turbid-amber, reaction alkaline, specific gravity 1.013, albumin none, sugar none.

Ocular Examination.—The vision of the right eye has always been defective, and with the best correction of a hyperopic astigmatism the vision was only $\frac{6}{150}$. The media were clear, the disk a vertical oval of good color; no abnormalities in the retinal vessels, but there was a large, shallow, probably congenital crescent, somewhat in the form of a posterior staphyloma, at the outer side of the disk.

Corrected vision of the left eye was $\frac{6}{20}$, the refraction being a hyperopic astigmatism. The sight had been defective for some months and had grown worse for the past three weeks. Ophthalmoscopic examination revealed decided punctate hyalitis, a nearly round disk, of somewhat grayish color; the scleral ring was broadened outward and slightly downward, somewhat in the form of the so-called congenital coloboma. The arteries were about normal in size, perhaps a little smaller than normal; the veins decidedly distended, particularly the upper temporal vein, which was swollen and tortuous. Lying beneath the retinal vessels, and crossing the entire length of the eyeground in a vertical direction, there was a broad area of greenish-white exudation, somewhat knobbed in appearance, over which were scattered numerous glistening masses, probably of cholesterin. This mass began about two disks' diameters from the optic papilla, and followed in general terms the sweep of the retinal vessels, particularly as they passed downward and outward, as well as upward. There was some fringing of pigment at the margins, and, particularly

on the disk side of the exudation in the macular region, scattered small areas of pigment. Beyond the main mass of the exudation, and separated by a narrow band of healthy retina, far out in the temporal periphery, there was a second mass of similar greenish-white exudation. (See Text-Plate XXVIII.) The general elevation of this exudate was about 2 D., but in some areas, particularly in the thick masses in the lower and outer periphery, the elevation was as much as 6 D. In places it partly covered the vessels and in one part there were some twisted vessels which may have been new formed.

The visual field of the right eye was practically normal for form, somewhat contracted for red and green (Fig. 1); that of the left eye normal on the temporal side, but with large wedge-shaped defect upon the nasal side (Fig. 2).

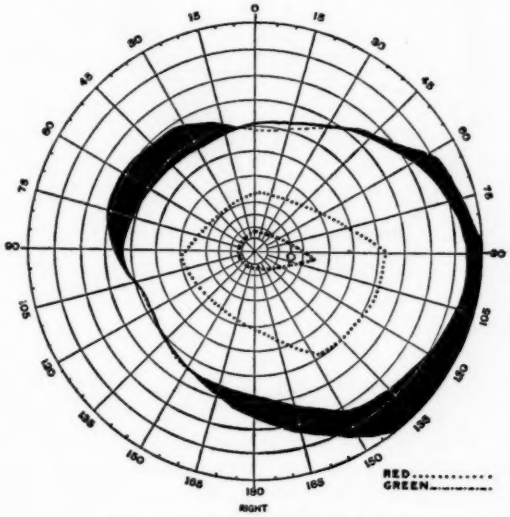

FIG. 1.—Visual field of the right eye.

At the suggestion of Dr. Riesman, the patient was placed upon a pill of reduced iron and arsenious acid, and later mercuric chlorid was added to the mixture. She also took Burnham's tincture of iodin, iodid of potassium, and, in order to demonstrate, if possible, whether the process was a tubercular one, received, both by my direction as well as the direction of Dr. Riesman, a course of old tuberculin injections, reaching as high as 5*mg.*, without producing the

slightest general or local reaction. These injections were continued for over a week.

The patient's physician writes, under date of April 24, 1909: "Her general health is good. I have examined the left eye with the pupil moderately dilated, and the glistening cholesterin masses seem to be less marked; that is, they are not piled up so high as they were when you examined

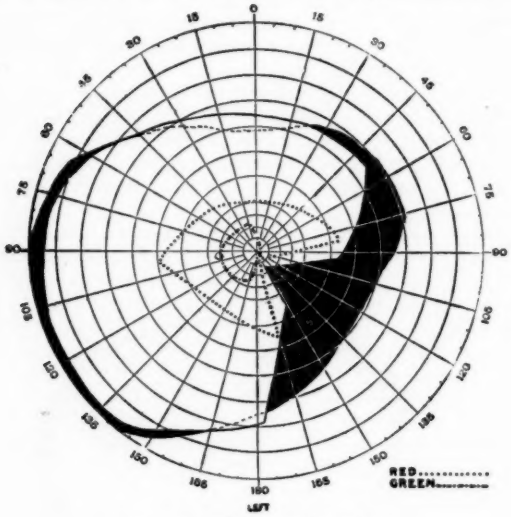

FIG. 2.—Visual field of the left eye.

her. On moving the eye numerous floating, glistening white bodies are perceptible, about the size and shape of oxalate of lime crystals as they are seen in the urine. I do not recall that these were previously present."

When I first examined her there was marked punctate hyalitis, and it is therefore possible that there has been some increase in this condition, giving rise to the appearance which her physician describes in the previous paragraph.

Remarks.—Dr. Coats has studied this disease in three groups, thus: Group 1, Cases without Gross Vascular Disease; Group 2, Cases with Gross Vascular Disease; and Group 3, Cases with Arterio-venous Communication. Referring to the first of these groups, he writes[1] concerning the case just

[1] Personal communication.

reported (he having examined the water-color which depicts the lesions in the patient's eye-ground, and the clinical record), "I should think your case undoubtedly belongs to the same class."

When the patient came under observation, the possible etiological factor claimed immediate attention, and at first it was thought that a tuberculous process was present, especially as in some respects the fundus lesions were similar to those described by W. Krauss and A. Brueckner.[1] As will be remembered these authors obtained a general reaction to tuberculin (0.5*mg* O. T.), as well as decided changes in the form of increase of retinal hemorrhages after each injection, in their first patient, who also had "suspicious signs of tuberculosis at the apices of both lungs." In their second patient, physical examination being negative, two injections of old tuberculin (*2mg*) gave no general reaction, but caused some changes in the fundus picture, *i. e.*, increase of hemorrhage and greater prominence of a layer of exudation in the summit of a detached retina. In one case reported by G. Carpenter and S. Stephenson in their list of cases of tubercle of the choroid, quoted by Coats and with some hesitancy included by him in the present group, the patient had tuberculous disease of the right knee and slight enlargement of the cervical glands. The patient of Paton whose excised eye was examined by Parsons, the case history and pathological examination also quoted by Coats, had enlargement of the cervical glands. The case is described as one of "tuberculous choroiditis" with a question sign, but the sections of the eye failed to reveal any lesions certainly characteristic of this infection. In Wagenmann's case, reported in connection with Von Hippel's record of "A Very Rare Disease of the Retina," which belongs to Group 2 of Coats's classification, there were signs of tubercle in the apex of the right lung, and the cervical glands were enlarged.

To quote the words of Coats, "perhaps the usual clinical diagnosis in cases of this kind is tubercle of the choroid, active or obsolescent." The pathological examinations thus far made are against this etiology, because, as Coats points out,

[1] Archives of Ophthalmology, xxxvii., No. 5, 1908, p. 544.

if the lesions were tuberculous they would have originally been situated in the choroid and of necessity destroyed it, while in his series this membrane was "always distinguishable and sometimes almost free from change." Tubercle of the retina is extremely rare, and usually involves the inner retinal layers, while in Coats's specimens of massive retinal exudation the outer layers were "chiefly involved." He regards the evidence from tuberculin reaction as "very equivocal," but admits that genuine tubercle could readily closely simulate the cases at present in discussion, and that therefore Krauss's and Brueckner's cases may have represented instances of tuberculous disease of the choroid.

Returning to my own case, there is nothing in the clinical and family history to suggest tuberculosis except that an uncle of the patient died of this infection; the tuberculin injections failed to produce the slightest general or local reaction, and, finally, there has been no increase in the local lesions up to the present time. Tuberculosis may therefore, I think, be eliminated from the diagnosis.

Nothing in my patient's family or personal history throws light on the etiological factor, inasmuch as the illnesses which she experienced bore no relation to the advent of the ocular troubles. Except for a slight anæmia, the general examination was negative. Neither hereditary nor acquired syphilis was present. It is interesting to note that the affected eye was the left one; usually, however, the right eye has been the one attacked; according to Coats, in the proportion of 11 to 6. In the cases thus far reported the male sex has preponderated (14 to 5). My patient was a young woman, whose age, however (nineteen), exactly corresponds to the average age worked out by Coats, if one doubtful case is omitted.

Exactly how long the ocular disease existed prior to my first examination, in October, 1907, cannot be stated, but the following quotation from a letter from her physician is of interest in this connection: "My notes show that on June 20, 1906, vision of the left eye was $\frac{5}{6}$. My next examination was on August 24, 1907, when vision had fallen to $\frac{1}{2\frac{1}{4}}$, and it was then I first detected the trouble in the fundus. In June of that year the patient had completed a four years' course at school in a distant city, and during the previous winter on one

occasion was seized with a very severe pain over the left eye and was sent to the hospital of the school, where it was necessary to use morphin. This pain continued for twenty-four hours. Whether it had anything to do with the trouble in the eye or not I do not know. The patient's mother noticed at Easter that her daughter's vision was failing."

It would therefore seem that the ocular disorder must have begun some time between the 20th of June, 1906, and the 24th of August, 1907, at which time its earliest stages were first detected. It is probable that they had already begun in Easter of that year, therefore about the end of March. It is stated that the first ophthalmoscopic lesion discovered consisted in retinal hemorrhages. These, however, were not manifest at the time she came under my observation. Very likely the period of time during which these extensive lesions developed was a greater one than would be apparent from the records, and Coats calls attention to the probable insidious development of the disease, and to the fact that it doubtless exists for a much longer period of time than many of the clinical reports would indicate.

Thus far treatment of the patient whose history I have reported has been unavailing, although there has been no progression of the disease, and, in fact, no notable alteration in the fundus lesions, and this record can do no more than place another example in the first group of Coats's classification of retinal disease with massive exudation.

There seems very little doubt that Groups 1 and 2—that is, those in which there is no association of gross vascular disease with exudation, and those in which such association exists—have the same pathological basis. Group 3—that is, those cases with arterio-venous communication—I have not referred to in the present paper, as I have not had the opportunity of studying this affection. Coats's pathological investigations indicate, as do also in most instances the clinical examinations, that while ophthalmoscopically the disease resembles tubercle, it is not of this nature, and he believes that these massive exudations are due to the slow organization of hemorrhages. Those who are interested will naturally consult Coats's paper for the full particulars of his pathological investigations.

In this connection I will report a second case, which probably, although not certainly, belongs to the same class.

Case 2.—A man, aged about sixty, came under observation November 16, 1907, in consultation with Dr. William J. Taylor, who has been good enough to furnish the following notes:

Family History.—So far as he is aware, there is nothing in the patient's family history which would throw light upon his ocular lesions.

Personal History.—The patient has always been, in general terms, nervous and apprehensive; for example, he was never willing to go about by himself, would not permit himself to remain within a room the door of which was locked, etc. He has manifested a gouty tendency, and has made several visits to Carlsbad on this account. In recent years he has exhibited a mild type of arteriosclerosis, although the arterial tension was never very high, nor the changes in his blood-vessels particularly marked.

General Examination.—The patient is a pallid, spare man, with slight stiffening of the radial vessels, but no definite cardiac disease. In point of fact, examination of the heart has always shown a regular action, the pulse being 75, full and strong. A blood examination, made April 14, 1907, was as follows: Hemoglobin 70%, erythrocytes 3,230,000, leucocytes 7,700. A number of urine examinations made between April 4th and the time that he came under observation revealed an average specific gravity of 1024, no albumin except on one occasion, and no sugar. Microscopically, there were numerous mucous threads, cylindroids, and a few hyaline and granular casts.

Ocular Examination.—The earlier ocular examinations were made by Dr. Charles A. Oliver, who has been kind enough to send the following communication: On December 3, 1906, the patient's eye-ground presented the characteristic changes of old ametropic choroiditis, which was more marked in the right eye. Correction of the ametropia, as follows:

R. E. — 2.75 — 0.75 c. axis 90
L. E. — 2.50 — 0.75 c. axis 90

yielded normal vision. On the 3d of May, 1907, the patient complained of "a spot" before the right eye. The fundus appearance was unchanged; a vitreous opacity was present; vision was normal.

On the first of June, 1907, "the spot" before the right eye was much better and vision continued to be normal.

Somewhat later the patient communicated with Dr. Oliver, and in this communication stated that his eye had become slightly clearer and that "the black element floating through it" seemed at times to "settle toward the nose." Practically no change was present when an examination was made sixteen days later, vision continuing to be normal.

At the next ocular examination, made by Dr. Oliver's assistant during Dr. Oliver's absence, the patient gave a history of rapid failure of vision, with neuralgic attacks and pain around the right orbit. The vision of the right eye was now reduced to $\frac{1}{15}$th of normal; vision of the left eye continued normal. The ophthalmoscope revealed obscuration of the disk, with much swelling of the retina, two spots of hemorrhage down and out; arteries narrowed veins large and full.

Somewhat later in the same month I examined the patient, with the following result: Vision of R. E. $\frac{1}{60}$, L. E. $\frac{1}{4}$. In the right vitreous there were punctate and weblike opacities, and a large area of white exudation was noted above the disk, in the macular region and down and in from the disk, where it assumed a yellowish-white appearance and was elevated 4 D. above the level of the unaffected eyeground, the mass appearing to be for the most part beneath the retinal vessels. The edges of the disk were cloudy; the veins uneven and full. In the left eye the conditions were exactly the same as those which have been already described in Dr. Oliver's report, namely, a moderate degree of epithelial choroiditis, without any signs of exudation or hemorrhage. After a long course of treatment, which consisted in the administration of iodid of potassium, nitroglycerin, and vapor baths, the vision remained unchanged in so far as the right eye is concerned and continued to be normal in the left.

At the last ophthalmoscopic examination, April 21, 1909, the exudation before described had assumed a somewhat knobbed appearance and was elevated as much as 6 D. above the level of the eye-ground in places, and showed a brownish-yellow color in various portions of it. The veins were dilated, and the arteries showed sclerotic changes, but there were no new vessels. Above the macula the exudation assumed a concentric shape. Tests to determine whether or not the lesion was tuberculous were not applied, but there was nothing in the clinical history or in the general examinations to indicate the likelihood of a tubercular infection. Syphilis can be excluded, and the whole process is best explained by assuming the presence of an exudation which had formed as a result of hemorrhage.

Remarks.—In some respects this case resembles the one reported by Walter H. Jessop, in the person of a woman, aged sixty-three, which Coats has included in his doubtful list. Jessop found about $\frac{2}{3}$ O. D. from O. D. an oval swelling covered by the retina, not transparent, about 3 O. D's in length and $2\frac{1}{2}$ O. D's in height. The edges were fairly distinct, the swelling of a grayish color and in places white and reticulate, retinal vessels crossed it, its greatest height was 4 D., and at one side there were some brown pigment patches.

If the case which I report should be classified with those now under discussion, it has one important and interesting feature, namely, that the eye-ground was the subject of expertexamination at stated intervals for a year prior to the greatest manifestation of the disease. Moreover, except vitreous opacities and ametropic choroiditis, there was no lesion in the patient's eye as late as four months prior to the beginning of the ocular affection, which, moreover, first appeared, according to the record of Dr. Oliver's assistant, in the form of a retinitis of considerable extent, with obliteration of the optic nerve edges and two small hemorrhages down and out from the nerve-head. Therefore, some time between the 1st of July, 1907, and the 20th of November, 1907, the process which I have described in its greatest elaboration must have taken place. Practically it has not altered during the time I have had this patient under observation—that is, from the 26th of November, 1907, until the 21st of April, 1909, or a period of seventeen months. At the last examination the patient was in excellent general condition and the affected eye entirely free from pain and inflammation.

NEURORETINITIS IN CHLOROSIS, WITH REPORT OF A CASE.[1]

By Dr. WM. CAMPBELL POSEY, Philadelphia.

(With two text figures and two drawings on Text-Plate XXIX.)

ALTHOUGH there are many subjective symptoms of chlorosis, it is comparatively seldom that structural changes occur in the fundi, just as it is rare to see extensive histological changes throughout the body the direct result of this disease. Thus, in a paper on the "Ocular Manifestations of Anæmia," which the author read before the Section on Medicine of the American Medical Association in 1897, it was noted that of 19 cases of marked chlorosis examined by him, appreciable swelling of the disk, or true neuritis, was not present in any instance, though a slight obscuration of the edges of the disk, with broadening and some pallor of the retinal veins, occurred in quite a number. Of 50 chlorotics examined by Saundby and Eales in 1882, however, some degree of neuroretinitis was found in 8%.

In addition to these slight degrees of neuroretinitis, chlorosis at times evokes a most intense inflammation of the optic nerve and surrounding retina, and a number of cases have been recorded where the changes in the fundi were so marked that they were mistaken for the ocular inflammations of albuminuria and of brain tumor. The writer has recently had a case of this intense form of neuroretinitis due to chlorosis under his care at the Wills Eye Hospital, the notes of which, together with an accurate picture of the inflammation when at its height in the two eyes, follow, the report being made

[1] Read before the American Ophthalmological Society, July 14–15, 1909.

with a view to calling further attention to this unusual form of ocular manifestation.

M. B., a Jewess, twenty-one years of age, was admitted to the wards April 10, 1909, complaining of poor vision and general weakness. The family history was negative regarding tuberculous, nervous or mental, or other serious disease, both father and mother living and well at the ages of fifty-four and fifty, respectively, while a sister aged thirty-six and a brother aged twenty-nine are both in excellent health. The mother had suffered no miscarriages and had borne no other children.

The patient was born in Russia, but has been six years in America. She attended school until fourteen years of age, since which time she has worked in factories as seamstress. Her hours have been long and she has had but a half hour for lunch. She has had no exercise, riding to and from her work, and reading incessantly in the evenings. She says her home is comfortable, and that she sleeps in a large, well-ventilated, and sunny upstairs room. She has never been very strong, but always fairly well, never having had to miss work on account of illness. She menstruated at thirteen years of age, always regularly, her periods lasting over three or four days, and being attended with but little pain. She has worn glasses for six years (high convex cylindrical glasses prescribed at the Pennsylvania Hospital), but had never had any previous ocular trouble beyond a transient inflammation in her left eye which the glasses corrected. She is not a mouth-breather and had never had any trouble with nose and throat until the past winter, when she began to suffer from frequent colds in the head. Her appetite has always been good, her bowels regular, never constipated, and she had never complained of shortness of breath or palpitation. There had never been any difficulty in urination. Any exposure to venereal contagion was denied. Her digestive powers were good. She was accustomed to drink one cup of coffee and two of tea daily, sometimes more.

Her present attack began about two months ago, when, in addition to repeated attacks of colds in the head she developed a general sense of discomfort and weakness. There was pain, nearly constant in the subclavicular region and axilla on the right side, which was not increased by deep breathing, but always made worse by changes in the weather. Her extremities were never cold or swollen. Headache was not complained of, but riding in the cars nauseated her somewhat. The day before admission she noticed that the vision in her right eye had become obscured,

apparently without cause and without pain, and the next day, on waking, finding the blur persisting, she decided to seek relief at the hospital.

On examination, it was found that the eyes presented nothing abnormal externally, save for a somewhat sluggish pupil on the right side. The ocular movements were normal. Vision in the right eye was reduced to counting fingers at 1 foot; corrected vision in the left equalled $\frac{6}{12}$.

As shown by the accompanying illustrations, which were sketched by Miss Washington a few days after admission,

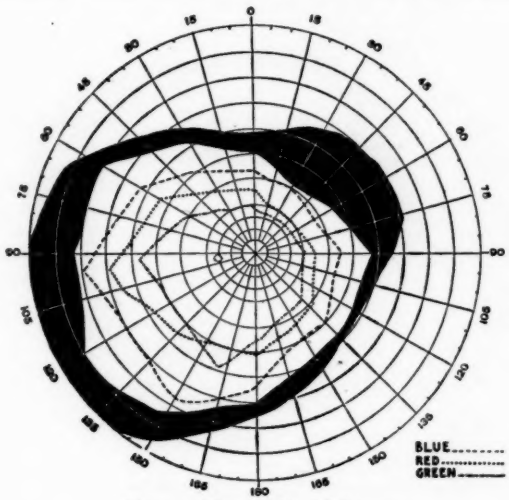

FIG. 1.—Visual field of left eye.

both eyes were the seat of an intense neuroretinitis, that of the right eye being accompanied by great œdema of the retina. The veins in both eyes were enormously distended, while the arteries in the right were much reduced in size. The arteries in the left eye were of about normal calibre. Both veins and arteries were tortuous, especially the former. No spontaneous venous or arterial pulse was demonstrable or could be elicited by pressure. The veins were dark and full and hidden in places by the swollen retina. There were no hemorrhages or isolated areas of extravasation in either eye save for a small, round, fluffy area of extravasation in the retina of the right eye far in the periphery in close association with the inferior temporal vein. The swollen veins could be followed into the periph-

ery of the retina, but the arteries in the right eye gradually faded away some 2 or 3 disk diameters from the papilla. The fields in both eyes were somewhat concentrically contracted; that of the left eye was free from scotoma, while a large absolute scotoma extended 20 degrees from fixation upward and outward in the right eye. (See charts.)

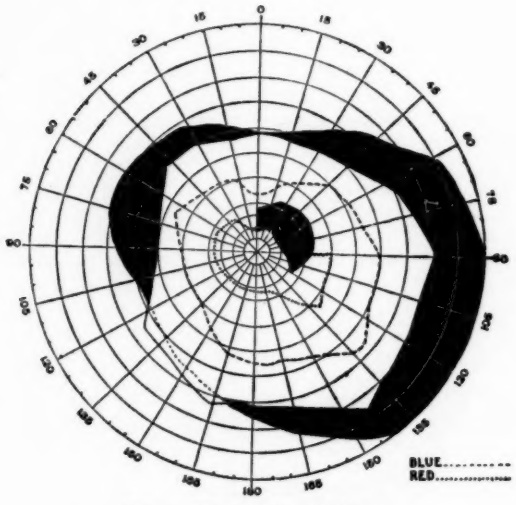

FIG. 2.—Visual field of right eye.

The unusual size of the veins awakening the suspicion of a profound blood dyscrasia, Dr. George W. Norris was called in consultation, and the writer is indebted to him for the following report:

Face and lips pale, finger-nails slightly curved and somewhat cyanotic. Hands cold. Body poorly nourished and developed. Right shoulder lower and smaller than the left.

Chest: Good expansion, equal on both sides. Jugular veins rather full when recumbent. Lungs: resonant throughout, except for small patch of dulness in the right axillary line above the hepatic dulness. Over this area vocal resonance and fremitus are diminished and the breath sounds weak. This region occasionally becomes painful (evidently pleural thickening).

Temperature 98°. Pulse 108. Respirations 20. Blood-pressure: systolic 120, diastolic 100*mm* Hg.

Blood: Hemoglobin, 60%; erythrocytes, 4,790,000; leucocytes, 11,100.

Heart: Slightly enlarged to the right. There is no thrill. The aortic second sound is slightly, and the pulmonic second sound greatly, accentuated. There is no arhythmia. The apex-beat is in the fourth interspace just within the mid-clavicular line. Slightly within the apex-beat a presystolic mitral murmur is heard, followed by a good muscular sound, and a short systolic murmur. Neither of these murmurs is transmitted.

Abdomen: Shows nothing abnormal. Neither liver nor spleen is enlarged. The stomach, according to auscultatory percussion, is normal in size and position. There is no rigidity or tenderness.

Diagnosis: Chlorosis. Mitral stenosis and insufficiency (slight).

Examination of the urine was negative.

Dr. F. R. Packard kindly examined the naso-pharynx and accessory cavities, but reported nothing abnormal.

In the absence of all other symptoms, it appeared that the ocular inflammation had been occasioned solely by the anemia, and the patient was accordingly placed on full doses of iron (Blaud pills, gr. v, t.i.d.) and rest, with some slight exercise in the open air enjoined.

A blood count made a week later showed: Hemoglobin, 65%; erythrocytes, 4,710,000; leucocytes, 9,600. Corrected vision in the right eye had also risen to $\frac{8}{60}$, that in the left remained at $\frac{6}{12}$. The eye-grounds too showed marked improvement, the retinal œdema and swelling of the disks having largely subsided, and the size of the veins being much reduced. The patient appeared in the best of spirits and said that she felt much better.

Hirschberg was the first (in 1879) to note the occurrence of optic neuritis in chlorosis. Other cases were reported shortly after this, however, by Bitsch (1), Gowers (2), Williams (3), Mackenzie (4), Schmidt[?] (5), Dieballa (6), and others, so that there are now about 20 fully recorded cases in the literature.[1]

Avoiding a detailed report of each of these cases, it may be briefly stated that in all a binocular inflammation of the optic nerve and retina of the most intense type was found to be dependant solely upon chlorosis, the reduction in the

[1] For literature consult Westcott and Pusey, ARCHIVES OF OPHTHALMOLOGY, 1902, p. 23; also Wilbrand and Saenger, iv., 1, p. 434.

percentage of hemoglobin in some of the cases being but comparatively slight, while in others it sank to a low degree—20%.

In all there was greater or less papillœdema, with obscuration of the edges of the disk, and more or less œdema of the retina round about the disk. In nearly all, mention is made of the great increase in the size of the veins, of their tortuosity, and of their disappearance at times in the swollen retina. The arteries were often very small, sometimes of normal size and usually tortuous. In a number of cases the general infiltration of the retina was replaced by the appearance of dots and streaks which arranged themselves at times in the form of a star about the macula, as is so frequently observed in albuminuric retinitis. In a case recorded by Schmidt (5), where there were a few small hemorrhages and sharply outlined white plaques of different sizes scattered over the retina, and a sprinkling of small glistening points and patches in a semicircular zone about the macula, three separate medical examinations were made, before albuminuria, cerebral disease, and hysteria could be excluded and the inflammation referred to its proper source, examination of the blood revealing hemoglobin 56%, red blood-corpuscles 120,000, white corpuscles 8,000.

In quite a number of other recorded cases, also, retinitis albuminuria was simulated, and the differential diagnosis only made by the positive finding in the blood and the absence of changes in the urine.

Small hemorrhages were noted in a fair proportion of the cases, usually in association with the larger blood-vessels. The most frequent and largest extravasations of blood into the retina in chlorotic patients occur, however, at the menstrual period, which Wilbrand and Saenger explain by the scanty amount of blood in the vessels at that time, and the decrease in the blood-pressure; in an interference with the circulation at the point of bending of the central vein, and the increase in size of the vein in consequence of the influence of the intraocular pressure; and, finally, in an abnormal transparency of the blood in addition to the presence of great local diapedesis.

Symptoms highly suggestive of brain tumor were present in many of the cases, and intense and almost unendurable

headache, with vomiting, dizziness, and in not a few cases localized and general convulsions, served in a number of instances to deceive for a time the physician in charge into the belief that a neoplasm was creating the symptoms. In one remarkable instance observed by Engelhardt (7), in which he was able to follow the case from early in its course until death, the mistake in the diagnosis was confirmed only at the autopsy, when it was ascertained that the choked disks, which had been followed by atrophy and blindness, the terrific headaches, and profound drowsiness, dizziness, and general convulsions, were all occasioned by cerebral anæmia without other pathological change. A tumor of the left cerebral hemisphere in the motor region had been suspected, as right hemiplegia of the extremities was present with total right hemianæsthesia. A blood examination made shortly before death showed hemoglobin 50%, erythrocytes 3,000,000, no increase in leucocytes.

Other observers also have experienced no little difficulty in properly differentiating the neuroretinitis of chlorosis from the papillœdema evoked by brain tumor, and Bannister (8) has reported[1] an instructive case where, but for the suddenness of the onset of the headache and other symptoms which suggested a doubt as to the existence of brain tumor and led to the examination of the blood, the diagnosis of brain tumor would have been established. In fact, the true nature of the case was only assured by the success of an iron and arsenic treatment. At the same meeting Patrick (9) referred to a somewhat similar case, in which, in addition to marked symptoms of cerebral irritation which pointed to a cerebral neoplasm, Wilder had found double choked disks with some small retinal hemorrhages and a paresis of the right external rectus muscle.

The association of paralysis of the external rectus muscle with neuroretinitis has been recorded in quite a number of the cases, *i. e.*, by Stölbing, Riegel, Remer, Dieballa, and others, Stölbing explaining the occurrence by the implication of the sixth nerve in the vascular sclerosis of the cavernous sinus, which he claims is present in all chlorotic cases.

Hawthorne (10) believes that the simultaneous occurrence of

[1] Amer. Neur. Assoc., May, 1898.

the inflammation of the optic nerve and the sixth-nerve paralysis suggests a common origin, the nature and temporary duration of the two events making it difficult to imagine any single cause capable of producing them other than an intracranial lesion, and, as intracranial thrombosis is a recognized possibility in chlorosis, he asserts that it may be concluded with considerable surety that it is the underlying cause in producing the double vision. While, as Gowers (11) has remarked, the evidence of intracranial thrombosis in most cases of chlorosis is very small, nevertheless it must be acknowledged that Hawthorne's hypothesis is not without authority, for no less an observer than W. H. Welch has pointed out that chlorosis must always be assigned a leading place among the causes of spontaneous thrombosis of the central veins and sinuses in women. Kockel, Pincles, and Leichtenstern have also observed thrombosis from chlorosis.

Ballaban (12), too, attributes the optic neuritis to thrombosis, but believes that the clot forms in the central vein, and not within a cranial sinus, basing his theory upon the study of a case in which there was intense neuroretinitis with veins three or four times the normal calibre. The arteries were very small. The blood columns in the veins presented a segmented appearance, and he was able to note the separation of the blood-current into colored and uncolored parts. Neither pulsation nor expulsion of the blood from the veins on the disk could be produced by pressure upon the globe with the finger. According to Ballaban, spontaneous thrombosis arises in the veins in consequence of the faulty condition of the blood and the poor circulation, especially since the latter may occasion disease of the endothelium of the veins. In the case observed by him the blood showed oligocythemia.

Dieballa (6) avers that any local and transient cause which increases the anæmia in chlorotic cases tends to produce papilloretinitis, and Schmidt (5) and he are agreed that the inflammation in the nerve is the result of a toxæmia, the toxic products being generated by deglobulization. The former thought that in a case observed by him two influences were at work in the deglobulizing process—a physical strain (dancing) and the sudden appearance of menstruation after a period of amenorrhœa. In his very interesting remarks upon this

subject, Dieballa goes on to say that he considers physical exercise in chlorotics to be decidedly deleterious for the blood, as it consumes oxygen, the supply of which is already lessened in consequence of the lessened oxygen-bearing hemoglobin. He has observed also that menstruation lowers the percentage of hemoglobin as much as from 8 to 10%.

Bannister sought the cause of the neuroretinitis in a cerebral irritation, the nature of which he thought still a matter of conjecture. He pointed out, however, that if the menstrual function is really in any way an excretory one, as some have held, or if the internal secretion of the ovary may become perverted or insufficient in such a way as to poison the system and produce chlorosis, it is not going much further to presume that it may in special cases cause symptoms strongly resembling those of organic cerebral disease and lead to embarrassing mistakes in diagnosis.

In the last volume of their exhaustive and invaluable work, Wilbrand and Saenger assert that, as Lenhartz has frequently found a very high intraocular pressure in patients with anæmia and chlorosis upon whom lumbar puncture was practised, they believe the papillœdema in chlorosis is occasioned by increased pressure as well as by the changes which occur in the vessels of the papilla. The changes observed in the vessels are explained by the lessening in the elasticity of the walls, by the abnormal constitution of the blood, and by fatty degeneration of the intima.

A recent paper by Cushing and Bordley (13), of Baltimore, tends to confirm this theory, although their remarks are directed to the solution of the problem of the origin of the cerebral and ocular changes observed in uræmia and albuminuria. These authors, after speaking of the generally accepted theory that the symptom-complex in nephritis is due to some toxic disturbance arising either in a disordered metabolism or in the retention of some products of normal metabolism which fail of elimination through the diseased renal epithelium, call attention to the suggestion made by Traube in 1861 that the symptoms in nephritis, so characteristic of an increase of cerebral tension, might actually be due to a cerebral œdema—a mechanical process, therefore—rather than to any autotoxic disturbance. They then assert

that any swelling of the brain, be it a general or a circumscribed process, necessarily leads to compression symptoms, the eye-grounds furnishing at an early stage an objective and sensitive index of the process, and aver that this is even more true of œdema than of states of increased cerebral pressure due to an encroachment on the intracranial spaces by a solid growth or an abscess. Their ultimate conclusion is that "the alterations of the eye-grounds in nephritis can be attributed in part, if not wholly, to the mechanical effects of increased intracranial pressure resulting from cerebral œdema." While they consider it a quibble to refer the œdema back to some toxic agency, as in a sense all œdemas are remotely due to toxic disturbances, they think that it is best to be conservative and say at least that "the symptoms are elicited by œdema resulting from some toxic agency, and are not, as is commonly supposed, due to the direct effect on the cerebral tissue of the toxic agent alone." They believe, further, from their clinical and experimental studies of choked disk, that the œdema of the retina itself is due in large part, even in cases of nephritis, to a backing of cerebrospinal fluid under increased tension into the investing meningeal sheath of the optic nerve.

While the writer is not in a position to combat this theory, it appears to him unlikely that increased intracranial pressure could have been a potent factor in evoking the changes in the eyes in the case which he has reported, for absolutely no other symptoms of cerebral irritation were present at any time during the course of the disease. He is inclined to attribute the changes in the eye-grounds primarily to the altered condition of the blood, but is at a loss to explain why such extensive and unusual changes should complicate an anæmia of so moderate and not uncommon degree.

Should it be established that the neuroretinitis of chlorosis as well as that of nephritis is occasioned by increased intracranial pressure, the recommendation of Cushing and Bordley, of "the propriety of permanent decompression in selected instances of renal disease when medical measures or lumbar puncture have failed to relieve existing cerebral (uræmic) symptoms, or when blindness is threatened owing to rapidly advancing degenerative changes in the neuroretinal tissues,"

will have to be taken into consideration in cases of anæmia as well as of nephritis. The influence of iron, however, upon the ocular changes in anæmia is very remarkable, and in nearly all of the reported cases vision which had been profoundly affected rose to normal, eyes which had been the seat of the most intense inflammation of optic nerve and retina regaining full acuteness of vision and an almost normal ophthalmoscopic picture under the administration of that drug.

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METASTATIC CARCINOMA OF THE ORBIT, WITH THE REPORT OF A CASE.[1]

By Dr. EDWARD A. SHUMWAY, Philadelphia.

(With a drawing on Text-Plate XXX.)

THE involvement of the orbital tissues and ocular muscles by carcinoma, as the result of metastasis from distant organs, without involvement of the eyeball, is very rare, only four cases being on record. Metastatic carcinoma of the choroid, with extension through the sclera to the orbit, is much more common. Since the report by Perl (1), in 1872, of the first case of metastatic carcinoma of the choroid, about 68 cases have been described. The literature of the subject has been carefully reviewed in recent years by Krukenberg (2), and Oatman (3), and especially by Suker and Grosvenor (4) in a paper last year before the American Academy of Ophthalmology and Oto-Laryngology. Suker and Grosvenor collected 64 cases, including one of their own and three unpublished cases seen by Ginsberg, Leber, and Sutphen. To these should be added cases by Bock (5), Schmidt-Rimpler (6), Pollack (7), and Lafon (8), a total of 68. In thirteen of these[2] there was an extraocular extension of the growth from the original focus in the choroid, but with few exceptions the orbital growth

[1] Read before the American Ophthalmological Society, July 14–15, 1909.

[2] Schultze, *Arch. f. Ophthalm.*, xxi., 1890; Uhthoff, *Internat. Beiträge z. wiss Med.*, ii., 1892; Holden, *Arch. of Ophth.*, xxi., 1892; Schultze, *Arch. f. Augenheilk.*, xxvi., 1893; Samelsohn, *Deutsche med. Wochenschr.*, p. 927, 1891; Michel, *Beiträge zur Onkologie des Auges*, Würzburg, 1899; Lagrange, *Tumeurs de l'œil*, i., Paris, 1901; Schmidt-Rimpler, *Lehrbuch*, 7th ed., Leipzig, 1901; Reis, *Klin. Monatsbl. f. Augenheilk.*, xli., 1903; Oatman, *Am. Jour. Med. Sci.*, 1903; Oeller, *Arch. f. Augenheilk.*, 1905; Thomson, *Arch. of Ophth.*, xxxv., 1906; Suker and Grosvenor, *Trans. Amer. Acad. of Ophthalmology and Oto-Laryngology*, 1908.

was small, did not interfere with the movements of the eyeballs, and was not diagnosed clinically. Cases of involvement of the intracranial portion of the optic nerves and of the chiasm have been recorded by Elschnig (9), Holden (10), Krohn (11), Dittrich (12), and Spiller and Weisenburg (13).

The five cases of involvement of the orbit and extraocular muscles are as follows:

Horner (14), in 1864, published a case of paralysis of the oculomotor nerve, with proptosis and severe headaches, in a man sixty-five years old. Four months after the first examination, all the recti muscles were paralyzed, there was ptosis of the upper lid, the eyeball protruded 3 lines, the pupils were dilated and immobile, the subconjunctival tissues were œdematous, the veins dilated and tortuous. No nodules were felt in the orbit, but growths appeared in the thyroid and in the submaxillary region. The eye later became so prominent that it could not be covered by the lids, and death occurred about six months after the patient was first seen. Post-mortem examination disclosed carcinoma of the dura at the base of the sphenoid, involving the right optic nerve at its entrance into the foramen, and extending backward and downward at the base of the brain. In the orbit nodules were found in all the recti and in the levator palpebræ superioris. Numerous nodules were present throughout the body: in the thyroid, sterno-cleido-mastoid and abdominal muscles, right pleura costalis, pericardium, peritoneum, and suprarenals. Horner thought the primary growth was in the dura, and that the first paralysis of the eye-muscles was due to pressure on the nerve, but that the subsequent proptosis and immobility of the eye were caused by metastatic nodules in the orbit and the muscles.

The second case was reported by Elschnig (15) in 1898. The patient, a woman seventy-three years of age, had carcinoma of the cervix, with metastases to the glands, skin, and right mamma; all of the eye-muscles in the left orbit were found to be the seat of nodules, with the exception of the inferior oblique. Microscopic examination showed hyaline degeneration of the muscle-fibres and almost complete degeneration of the nerves entering the muscle. Elschnig raised the question whether the disturbance in motility was due to the degeneration of the

nerves, or to the destruction of the muscle tissue and prevention of muscular contraction.

In 1899, Wintersteiner (16) described a case in a woman fifty-eight years old. The clinical history, recorded in one of the medical wards of the general hospital in Vienna, noted a disturbance of the motion of the right eye, which was ascribed to paresis of the internal rectus muscle. There was carcinoma of the left breast and neighboring lymphatic glands. Post-mortem examination showed metastases in the liver, retro-peritoneal glands, and of all the ocular muscles of the right eye with the exception of the inferior oblique. A careful microscopic examination of the entire orbital contents showed, in addition, a small nodule in one place near the ophthalmic artery, but no involvement of the optic nerve. The metastasis preserved partly the fibrous character of the primary growth, and had caused simple atrophy of the muscle tissue. The fibres were thinner, distinctly fibrillated, and the Cohnheim fields, which are very clearly marked in the intact fibres, were absent. The contractile substance had retracted, and the elastic sheaths surrounding the fibres had almost entirely disappeared, where the atrophy was evident. In no place was there any evidence of compression of the nerves or degeneration of the nerve-fibres, although here and there the cells followed them and were spread out in the perineurium. As the involvement of the muscles was a very early one Wintersteiner believes that his case answers Elschnig's question and proves that the disturbance of motility is due to destruction of the muscle tissue, and not to compression of the nerve. He thinks, too, as only one muscle was clinically paralyzed, although all but the inferior oblique were affected, that similar cases of involvement of the ocular muscles in metastatic carcinoma may not be so rare as has been thought.

The fourth case was reported by Axenfeld (17) before the Ophthalmologische Gesellschaft at Heidelberg, in 1907. The patient was a woman who, two years after extirpation of a mammary carcinoma, showed progressive paralysis of the muscles of the left eye. As there were no distinct orbital signs of tumor, it was thought at first that the paralysis was of nuclear origin, perhaps of a toxic nature. Subsequently,

exophthalmos appeared, and when the patient was seen by Axenfeld the eyeball was almost immovable, both actively and passively, and a firm resisting mass was evident above and below the eye within the orbital margin. The patient complained of severe neuralgic pain of the first and second branches of the trigeminus, and choked disk was present. The accessory sinuses were free, and as no other metastasis could be found in the body, intraperiosteal exenteration of the orbit was performed, and the bones covered by Küster's method. Primary union was obtained. Microscopic examination showed a conversion of the orbital tissue into a dense fibrous mass, with scattered nodules of carcinoma cells In this dense fibrous tissue the single muscles were distinctly recognizable as tubes, densely infiltrated with cells, which indicated that they themselves were metastatically involved. Axenfeld thought operation was indicated only in the extremely rare cases in which no other metastases could be found in the body. Even in such cases it was not always possible to be sure that they were not present.

In the discussion of Axenfeld's paper, Wintersteiner (18) referred to a case of metastatic sarcoma of the external rectus muscle in a woman who had been operated upon for myosarcoma of the intestines. Metastatic sarcoma of the orbit has also been described by Poliagni (19); and de Schweinitz and Meigs(20) reported a case of metastatic sarcoma of the choroid and orbit, in which the primary growth was found in the mediastinum. Axenfeld (21) thinks the latter case was probably of the same character as those recorded by Oxley, (22) Powell (23), and Tomasi Crudelli (24) as cases of malignant lymphoma originating also in the mediastinum.

My own case is as follows:

M. S., female, aged forty-nine years, was seen December 23, 1906, in consultation with Dr. I. W. Hollingshead, of Philadelphia, under whose care she had been for about ten days. An accurate history was difficult to obtain, as the patient was mentally confused, partly because of exhausting pain, and partly as the result of drugs administered to control it. She had noticed a disturbance of vision in the right eye for five months. In September, 1906, her glasses had been changed by Dr. P. N. K. Schwenk, who subsequently wrote me that her vision at this time, with a glass

correcting her hyperopic astigmatism, was $\frac{20}{30}$ in each eye, and no fundus changes or disturbance of motility of the eyeball were manifest. Shortly afterward she had an attack of pleurisy on the left side, and later developed some spinal trouble, with a gastro-intestinal disturbance. From this time on she was confined to bed, suffering with abdominal pain, nausea, and marked constipation, and her weight had gradually fallen from 165 pounds to 123 pounds. For three weeks the right eye had been very painful and prominent. On examination it was found to have no light perception, was proptosed straight forward about 8*mm*, and its movements were limited in all directions. There were intense bulbar injection and chemosis, the cornea was ulcerated, and the anterior chamber one-third filled with a yellowish-white exudate. The patient complained of intense ciliary tenderness and great pain, which could be controlled only by hypodermic injections of morphin. No history of a growth could be elicited, and a diagnosis was made of acute panophthalmitis, possibly due to metastatic choroiditis from some focus of infection in the pleural or abdominal cavity. Exenteration of the eyeball was therefore advised, and she was admitted to the University Hospital, where the operation was performed on the following day. Much to my surprise, although the anterior chamber was full of pus, the vitreous was found clear, and a nodular mass could be felt in the orbit back of the globe.

As I had not obtained permission for anything beyond the exenteration of the eye, the orbit was not explored further. On the next day there was still marked chemosis, which gradually subsided under the use of cold compresses, and on its disappearance the nodular mass could be felt extending forward nearly to the orbital margin, above and below. Further examinations by Drs. John Luther and A. O. J. Kelly showed typical scirrhus carcinoma, with retraction of the left breast, involvement of the axillary and supraclavicular lymphatic glands, fixation and enlargement of the uterus, thickening of the utero-sacral ligaments, and an obscure mass in the abdomen, in the position of the transverse colon, possibly retroperitoneal. On account of the evident wide-spread carcinomatosis, further operative interference was considered inadvisable, and commencing on January 6, 1907, the patient was given X-ray treatment by Dr. Henry K. Pancoast. The exposures, lasting from twelve to twenty minutes, were made on alternate days to the orbit and abdomen. At the same time injections of trypsin were made daily, beginning with 10 minims, and increasing 5 minims each injection until a dose of 60 minims was given. Under this treatment, and general tonic

medication, the orbital mass apparently disappeared. The breast remained unchanged and the patient's weight gradually declined, being 118 pounds on February 8th.

On January 21st she complained of considerable pain in the head and blurring of vision of the left eye. Ophthalmoscopic examination showed beginning optic neuritis, which was thought to be due either to involvement of the meninges or to general toxemia. On February 1st, after the injection of trypsin, the patient developed toxic symptoms, with a chill, rise of temperature to 101.6° F., and repeated vomiting. The site of the injection became indurated, and at this and another position large abscesses developed, and the injections were discontinued. On February 8th injections of amylopsin alternately with the trypsin were resumed, but after ten days were stopped at the request of the patient, because of the pain. The X-ray treatments were, however, continued until she left the hospital, March 30, 1907. At this time no growth could be detected in the orbit, and the growth in the breast and the axillary glands were reduced in size. She had steadily lost strength, however, was much emaciated, and there was almost complete anorexia, with frequent attacks of vomiting. Throughout her stay in the hospital the constipation was marked.

After reaching home she rapidly grew worse, the abdominal symptoms growing more pronounced, and died from intestinal obstruction one month later, April 30, 1907.

An autopsy was made on the following day by Dr. Samuel Leopold, and a diagnosis was made of scirrhus carcinoma of the breast, broncho-pneumonia, chronic adhesive pleuritis, chronic parenchymatous nephritis, fibrosis of the liver, chronic interstitial splenitis, and chronic peritonitis (multiple serositis). Sections were prepared in the McManes Laboratory at the University of Pennsylvania from the following tissues: mammary gland, spleen, liver, pancreas, kidney, uterus, ovary, broad ligament, and pelvic peritoneum. These were carefully reviewed by Prof. Allen J. Smith and a revised post-mortem diagnosis was made by him of scirrhus mucoid carcinoma of the mammary gland, with infiltrating, non-encapsulated metastases in the liver, spleen, kidneys, uterus, broad ligament, ovary, and pelvic adhesions. Dr. Smith's report is as follows:

(1) *Mammary Gland* (left): A section from this indurated breast shows, in addition to a few scattered and

small lobules of adipose tissue, the rest of the specimen to consist of a dense stroma of fibrillar connective tissue, in which are found occasional remnants of the old glandular structure and at places definite carcinomatous infiltrations. The fibrous matrix is of the wavy type characteristic of the mammary gland. A small bit of the epiderm overlies a part of the section. For the most part it is thin and evidently atrophic, but at one position extends in an irregularly branching root well down into the mass of the general stroma, this root inclosing a comparatively large cystic space lined with squamous cells and containing a loose concentric nest of the same. In the section there are to be found a very few atrophic gland lobules, showing no malignant change and several ducts without material alteration. Widely but sparsely scattered through the stroma are small groups of cells and isolated cells of glandular type of epithelium, some of which present the "seal-ring" appearance of so-called "colloid" change (really mucoid, the cells in reality the same as "goblet cells"). Examples of large and hyperchromatic nuclei are occasionally seen; but no examples of karyomitosis were encountered; and many of the cells are decidedly small and evidently atrophic. Several small epithelial-lined cysts also exist in the section, but present no appearance of malignant change.

(2) *Spleen.* The capsule of the organ is slightly thickened from fibrosis, and on the outer surface are met scattered strands of fibres and large endothelioid cells, probably representing the adhesive formations above mentioned. There is no appreciable overgrowth of the trabeculæ of the organ or of the blood-vessel walls; but there is a diffuse and at places a marked fibrosis of the pulp of the spleen. The section examined shows the vessels and capillary spaces filled with blood and irregular foci of hemorrhage both in the subcapsular position and in the interior of the organ. All through the section, especially beneath the capsule, and more concentrated at some positions than at others, there are epithelial cells of the same type as met in the carcinomatous breast. Mitotic figures are not infrequent and the cells, as a rule, are larger than those of the breast; here, as in the breast, examples of mucoid "seal-ring" cells are met. In no position in the section are these cancerous metastases encapsulated, but represent a diffuse and irregular infiltration of the splenic tissue. Numerous coarse, yellow, granular deposits of hemosiderin are met in the tissue. The Malpighian bodies, as a rule, are small and normal in appearance, but now and again are invaded by the cancerous elements.

(3) *Liver.* The capsule proper is not thickened, but beneath the capsule in several sections examined there are areas of replacement of the liver tissue by a diffusing non-encapsulated scirrhus cancer of the same type as above described. In this in places there are persisting bile-ducts, with normal epithelial lining, and occasional small deposits of golden granular pigment probably mark the sites of previously existing pigmented hepatic cells. In the cancerous areas of the liver there are noted particularly (seen in the previously described sections, but not so clearly and not nearly so frequently) fine examples of cell inclusions in many of the neoplastic cells. These are occasionally seen, too, in hepatic cells at some distance from the cancerous areas. They have more similarity to protozoa in their best examples than is common; being sharply defined by a clear, deeply eosin-stained wall, their interior granular or reticular and faintly stained by the hematoxylin, and in the centre a clear, hyaline, deeply eosin-stained, nuclear-like body. These inclusions vary in size, are spherical or ovoid in shape, and, while usually single in the cells, are sometimes present to the number of two or three. In addition, many of the cells show the usual seal-ring appearance with the hematoxylin-tinted mucin in the interior. The perilobular connective tissue varies. Usually of normal amount, it at places is distinctly and even greatly increased; and in such situations there is usually at least a minor presence of the infiltrating cancer cells. There is at no place in the section examined any attempt at definite encapsulation of the cancer foci. The hepatic cells at the border of the growth are commonly degenerate, often atrophic; but there are many cells, apparently hepatic in nature, which are decidedly approaching the appearance of the tumor cells and might well be appealed to as evidence of metaplasia. The general hepatic structure shows considerable venous dilatation, especially of the central lobular venules, and the columns of liver cells are widely separated and often broken as if the tissue were œdematous. The cells are more or less necrosed, and the remaining ones are granular and stain with poor definition.

(4) *Pancreas.* No important change in the vessel walls but some evidence of a venous hyperemia. Distinct overgrowth at places of the interlobular connective tissue and more diffused intralobular fibrosis of fairly marked grade. Islets of Langerhans for the most part intact, but some walled off by a thick and dense fibrous capsule and some the seat of intrainsular fibrosis. Scattered areas of post-mortem necrotic change evident. Otherwise parenchyma

in fair state. No cancerous metastasis recognized in material sectioned.

(5) *Kidney.* Two different sections examined, presumably from the two organs. Both show no appreciable change of the capsule, but the existence of minor fibroid change of the capsules of Bowman and local areas of intertubular fibrosis in the cortex. In both there is more marked fibrosis of the intertubular tissue of the pyramids. Both show advanced cloudy swelling and even necrosis of the cortical epithelium, with less marked change of the medullary parenchyma; no important desquamation; numerous casts in the tubules. In neither of the sections were there present the changes corresponding to the pale infarct noted in the gross protocol. But both show minute foci of infiltration in the cortex of cells of the same type as described in the neoplasms found in the previously mentioned organs. These were not encapsulated, but occurred in situations about vessels or Malpighian bodies where there was evident some increase of fibrous tissue in which the cancerous elements occurred.

(6) *Uterus.* Endometrium absent from sections examined, which include only a part of the thickness of the muscular wall of the uterus. The tissue is diffusely infiltrated by great numbers of the cancer elements already described, the cells large and well preserved, often hypochromatic or mitotic, and frequently showing the mucoid changes and the cell inclusions already mentioned.

(7) *Ovary.* The tissue is markedly sclerosed, without persistence in the section of Graafian follicles or other structures characteristic of the organ, save a number of dense and hyaline corpora fibrosa, the scars of former corpora lutea. Widely diffused through the organ are the same cancerous elements as already described in the organs above detailed.

(8) *Broad Ligament.* A section of the thickened broad ligament shows the presence of a diffuse cancerous infiltration of the same type as mentioned elsewhere; there is marked hyperemia and evidence of progressive connective tissue of the part.

(9) *Adhesive Material from Pelvic Wall.* This is merely a thickened fibrous structure containing a few strands of smooth muscle and diffusely infiltrated, as described for the other structures, with cancerous elements.

At the autopsy the roof of the orbit was broken through, and a hard nodule 1.5 by 1 by 1*cm* in diameter was found directly back of the remains of the eyeball, and was removed for

microscopic study. Sections were prepared by Dr. C. M. Hosmer in the ophthalmological laboratory. They include a cross-section of the optic nerve at the entrance of the central vessels, surrounding orbital fat and connective tissue, a portion of the inferior rectus muscle, and of either the internal or external rectus muscle. The orbital connective tissue is converted into a dense, fibrous tissue, which is diffusely infiltrated with epithelial cells of the same type as those described in sections of the mammary gland, liver, etc. The dense, fibrous tissue is either the result of a fibroid degeneration of the carcinoma metastasis, as described by Axenfeld in his case, or of the action of the X-rays. At the point of entrance of the vessels into the optic nerve the carcinoma cells follow the vessels through the dural sheath and surround the pial envelope as a ring, several cells deep. There is no invasion of the nerve-fibres, and sections a short distance back of this point show no involvement of the nerve at all. The nerve-fibres are in a state of advanced degeneration, with fine vacuoles in the spaces between the septa, ordinarily occupied by the axis-cylinders. The nerve is shrunken, considerable space existing between the pial and dural sheaths, and the septa within the nerve appear relatively broad, because of the disappearance of the nerve-fibres.

The two recti muscles exhibit marked infiltration of their substance with the carcinoma cells. In places the muscle bundles have been replaced by them, in others the cells have proliferated between the bundles. The remaining muscle fibres show an advanced stage of simple atrophy, with loss of the transverse striations and indistinctness of the muscle columns in cross-section. The infiltration is fairly well shown in the accompanying sketch by Mrs. J. D. Z. Chase (Fig. 1), in which the nerve-bundles appear especially prominent on account of the atrophy of the muscle substance.

The histological examination, therefore, confirmed the clinical diagnosis, and in the absence of involvement of the choroid, as proved by the examination of the eyeground by Dr. Schwenk, we are justified in placing it among the very rare cases in which the metastatic elements enter the branches of the ophthalmic artery which supply the muscles or orbital tissue, instead of passing into the more numerous ciliary

arteries, under which circumstances we have the production of the much more common condition, metastatic carcinoma of the choroid. Whether, as Wintersteiner suggests, the occurrence of orbital involvement is more frequent than has been assumed, is a matter for still further study. In every instance of isolated paralysis of an ocular muscle in an adult, the patient should be examined for carcinoma, and a metastatic nodule should be suspected in the presence of general carcinomatosis. The prognosis is very bad, and operation is only justifiable where metastatic growths elsewhere can be excluded, or where it is to be performed for the relief of pain.

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ELEVENTH INTERNATIONAL CONGRESS ON OPHTHALMOLOGY, NAPLES, APRIL 2–7, 1909.

REPORTED FROM MANUSCRIPTS BY DR. W. CLAUSEN, BERLIN.

Translated from Vol. LXIII., No. 3, German edition of these ARCHIVES, by Dr. PERCY FRIDENBERG, New York.

JOINT SESSION with the International Typhlologists. Pres. Prof. VITALI.

Discussion on the Theme: Is it advisable, in case of disease leading to blindness, to acquaint the patients with the prognosis, without reserve, in order that they may, while sight still lasts, practise some occupation which will suit the new conditions?

Report of Prof. NEUSCHUELER (Rome) urges that ophthalmologists, as well as pedagogues and philanthropists should pay more attention to the study of blindness and its psychology, as surgeons do to orthopedics. There are various criteria, social, educational, and medical, of "blindness," and no exact international standard in any category. NEUSCHUELER defines blindness, socially, as the condition in which reading is impossible with correcting glasses. In iritis, keratitis, retinitis, and so on, it is better to acquaint the patients with the probable results for vision, in order that they may prepare themselves. This must be done with humanity and tact, gradually if necessary, and in accordance with the requirements of the individual case. Instead of wasting time in useless treatment, the blind alphabet could be taught. In high myopia, too, this study would be very useful, but the ophthalmologist must study the subject of typhlology.

Discussion: ANGELUCCI supports the views of the reporter. DUFOUR (Lausanne), COLUCCI, ROSA MAYER, COZZOLINI, KOUBLY (St. Petersburg), DENTI, and HOOR (Buda-Pesth)

think it inadvisable to let the patients know of their fate while they can still see, especially as typhlologists find that it is much easier to instruct them after they have become quite blind, and the patient will have no confidence in the hopeless prognosis as long as he can see. Aside from the possibility of mistakes in prognosis, humanity will prevent our depriving the patients of hope.

First Scientific Session: Pres: De La Personne.

Uniform Estimation and Notation of Acuteness of Vision. Report of the Committee, (C. Hess, Chairman): Charpentier, Dimmer, Eperon, Jessop, Nuel, Reymond.

A test card with suitable numbers and Landolt rings is preferred to letters. The unit is that degree of acuteness in which two dots on a white ground seen at an angular distance of two minutes are distinguished as separate. The scale should be graded in tenths, from 1.0 to 0.1 and at 1.5 and 2.0 in arithmetical progression, to be read at a distance of 5 metres. Good daylight should be used in testing.

Discussion: Landolt (Paris) discussed the optical theory of ring-test objects.

Armaignac regrets that the rings, representing a minimum separabile, were used instead of a minimum visibile. Letters are preferable to numbers on account of their variety. Two numbers in a row are not enough. Snellen's pot-hooks are the best international test, as they can be used not only for illiterates, analphabets, and children, but for deaf-mutes, as well. The graduation of the committee's test card is faulty.

Ovio (Modena) objects to the minimum separabile, as it depends so much on light, form, space, and memory. Arithmetic progression is advisable.

Vignes (Paris) considers geometrical figures the only scientific objects for a uniform test. The 1.5 and 2 of the committee scale are superfluous.

Salzer (Munich) welcomes the use of letters and numbers. With geometric figures alone, we may underestimate the real acuteness of vision.

Grosz (Buda-Pesth) moves to adopt the Committee's test card.

Pfalz (Duesseldorf) finds Landolt's rings unsuitable for children and imbeciles. The geometric progression is the

better. Pot-hooks are most easy to recognize and should be included in the scale.

The suggestions of the Committee were adopted.

1. **New universal test-letter scale.** TOMAS BLANCO (Valencia). BLANCO presents a test card of black squares, especially for illiterates.

OVIO (Modena) prefers the Landolt rings to these squares. The angle of 1 minute, suggested by Blanco, is too small. It should be twice as large, for signs and numbers, as for letters.

2. **Correcting glasses in general pathology and the method of determination.** E. E. BLAUUW (Buffalo). Clinicians do not sufficiently recognize the importance of correcting ametropia. Vague and obstinate symptoms may be caused by lack of correction, especially in nervous patients. The use of a mydriatic is unnecessary, unscientific, and objectionable.

3. **Experiments on visual acuteness and the minimum separabile, with optotypes; accommodation and stereoscopic sense in monocular vision.**

E. MARRI (Turin) finds the angle of 1 minute too large as the last line of most test cards can be read at twice the estimated distance. The field of vision for a single eye is at first smaller than the binocular field, but this enlarges with practice, and stereoscopic vision also rises to normal.

4. **Elementary visual tests.** M. W. PERGENS (Maeseyck) presents 13 test cards with geometric figures with which he claims to be able to differentiate various internal diseases of the eye.

5. **Translucency of various opaque media of the eye as determined with a simple loupe.** TRANTAS (Constantinople) uses, instead of an electric transilluminator, a condensing lens of + 20 D. to + 30 D. On oblique illumination some light penetrates the sclera, giving a slight glow. In this way he was able to confirm the diagnosis of intraocular melanosarcoma in two cases.

6. **Formula for the visual field.** E. LOPEZ (Havana) suggests a new formula for the notation of the field of vision.

7. **The binocular field of vision after squint operations.** WOELFFLIN (Basel) used an illuminated perimeter and found that the outer halves of both visual fields were supplied by the corresponding halves of the retina, but that in the region of

the point of fixation double images were seen, close together, which could not be fused for several days. Repeated perimetric tests aid in the fusion.

Discussion: GONIN (Lausanne) confirms Woelfflin's observations.

8. **The notation of the axes of astigmatism and of perimetric charts.** A. S. PERCIVAL (Newcastle) advises beginning with the zero point at the right end of the horizontal meridian, seen facing the patient, and counting from right to left, *i. e.*, against the clock, in notation of cylinder axes and meridians of the visual field.

9. **Measurement of the lifting power of the adductors and abductors.** L. HOWE (Buffalo) describes his method of ophthalmo-dynamometry with actual weights and indicator scale, which he believes of practical importance in the choice of tenotomy or advancement.

10. **Oculistic instrumentarium constructed on rational principles.** LANDOLT (Paris) claims that the handles of all eye instruments should be made of ivory or aluminum. The form should be rectangular with the long diameter parallel to the axis of the plane of the blade or edge, and square in those instruments which have to be turned. The edges must be rounded and ground off. The instruments should not be too long, as is generally the case.

11. **Astigmatism.** M. MARQUEZ (Madrid) notes that there is lenticular as well as corneal astigmatism, and that the former should always be borne in mind. It can be determined by retinoscopy. Two cylinders may be needed.

12. **Requirements and regulation of signalling by color.** C. A. OLIVER (Philadelphia) considers his wool tests the best for general clinical purposes. For signal service the actual conditions must be duplicated. He has devised a test lantern for this purpose. There should be an international standard for the colors and regular inspection.

13. **Changes in the conjunctiva after exposure to light of short wave length.** A. BIRCH-HIRSCHFELD (Leipzig) experimented on rabbits with Schott's uviol lamp and produced simple catarrh, leading later to changes in the epithelium and vessels, with sub-epithelial proliferation and pigmentation. The pre-tarsal connective fibrillary tissue loses its

structure, becoming homogeneous and glassy. Hyaline degeneration of the vessel-walls and proliferation of the intima are noted.

Discussion: AXENFELD (Freiburg) notes that these experiments do not represent the conditions in vernal catarrh. It is a question whether these changes occur unless the conjunctiva is artificially exposed by ectropionizing, and whether there are any characteristic changes in the palpebral conjunctiva.

SCHANZ (Dresden) notes that vernal catarrh must be due to ultra-violet rays of less than 300 micro-millimetres, as only such are present in daylight at ordinary altitudes. This corresponds to the measurements found by Birch-Hirschfeld in his artificial light.

CRZELLITZER (Berlin) noted typical electric conjunctivitis in workmen engaged in electric welding. Schott's glass with high refractive index, and ruby glass, were tried for protection without effect.

GONIN (Lausanne) notes that vernal conjunctivitis is less common in the mountains than in the lowlands, so that the prolonged effect of ultra-violet light cannot be a causal factor in development.

TRANTAS (Constantinople) notes the dependence of vernal catarrh on sunlight, as shown by its variation directly with the temperature and climate. The cornea is rarely affected, and proliferative changes in the conjunctiva, like those noted in Birch-Hirschfeld's experiments, are seldom seen.

STEINER (Rome) thinks that Birch-Hirschfeld's experiments throw light on the whole subject of ophthalmia due to radiations. In closing, BIRCH-HIRSCHFELD disclaimed identifying the changes found by him with clinical vernal catarrh, but the cause is the same in both. When rays of less than 330 micro-millimetres are excluded by suitable glass plates, the uviol light has no effect.

14. (a) **Is there an amblyopia from non-use?** J. GONIN (Lausanne) notes that corneal opacities or refraction error of one eye disturb vision much more than when they are bilateral. This cannot be explained on purely optical grounds, and is the expression of functional weakness of

one eye due to partial exclusion from the visual act. The hypothesis of amblyopia ex anopsia is founded on fact.

Discussion: LODATO (Palermo) recalls his experiments on newborn dogs and cats in which, by tarsorrhaphy, he was able to cause amblyopia and arrest of development in the closed eye.

HOOR (Buda-Pesth) notes that amblyopia may be due to squint, although it does not follow that all squinting eyes are amblyopic, or that it is always due to deviation.

GONIN, in conclusion, had in view the recent work of Silex denying the existence of an amblyopia from non-use.

14. **(b) Curative effect of subconjunctival aseptic abscess in post-operative infection.** S. TERENTINOS (Patras) injects biniodid of mercury and petroleum in vaselin under the conjunctiva with good effect in ulcus serpens and in post-operative infections. The abscess acts curatively by producing antitoxins and is to be recommended in infected penetrating wounds and sympathetic inflammations.

15. **Plan for a uniform nomenclature of the motor anomalies of the eye.** A. DUANE (New York) notes that Greek and Latin are mixed in the present terminology and suggests rational uniform terms.

LANDOLT (Paris) notes this discrepancy in other fields of ophthalmology but advises the retention, for the present, of the traditional terms.

HOWE (Buffalo) thinks the terminology now in use can not be altered at once without some trouble and confusion. He advises the appointing of a Committee on Nomenclature to report at a later congress.

16. **Ocular injuries from a medico-legal point of view.** F. BARAZZA (Buenos Ayres) thinks special laws should be passed for industrial injuries, and that the loss of vision must be brought into relation with the injury. The workmen often refuse treatment or are negligent in following orders, and the results are not as good as they should be. Certificates should be made by oculists only.

17. **Visual efficiency.** A. PERCIVAL (Newcastle) presented various formulæ for an arithmetical calculation of damages for ocular injury, and two percentage tables for estimating loss of wage-earning capacity.

18. **Complete lymphangiectasis of the conjunctiva during pregnancy.** M. MENACHO (Barcelona) reports a case of this nature in which the chemosis disappeared completely after delivery, showing a reflex action between the uterus and the eye.

19. **New views on pathogenesis of primary cataract.** N. SCALINCI (Naples) with BOTAZZI made chemico-physical tests of the lens. It is formed of organic hydrogel, a combination of two colloids, soluble and insoluble phako-protein. Imbibition causes diffusion of the soluble protein and clouding, with disgregation of the fibres and of the insoluble protein. The lens colloids are alkali-proteins. Acids cause a precipitation and cataract is the result, as in acidosis of diabetes, uræmia, oxaluria, tetany, and in other disorders of metabolism. Glass blowers' cataract is due to carbonic acid accumulated in consequence of venous stasis, which may also act indirectly by causing changes in the vessels and ciliary epithelium.

Discussion: PARDO (Modena) asks for an explanation of unilateral diabetic cataract, and whether experimental cataract ever develops in a saline aqueous.

SCALINCI, in closing, explains unilaterality of diabetic cataract as due to differences in the alterations of the ciliary epithelium and vessels. Acid or alkaline aqueous may be present in cataract. Organic acids, which are weaker, produce marked changes. The investigations of Angelucci, Ovio, and Pardo deal mainly with osmosis and not with the physico-chemical composition, particularly their colloid nature.

20. **The cataract operation.** E. VALUDE (Paris) praises irrigation of the capsule-sac as the best means of removing cortical remnants which later on interfere so much with sight. Extraction in the capsule is not generally advisable. To prevent iris prolapse and make irrigation free from danger he advises corneal suture (with fine round needles and sewing-machine thread), which does not include the entire thickness of the membrane.

Discussion: MARQUEZ (Madrid) thinks a corneal suture objectionable in cataract operations except where there are marked gaping of the wound and danger of prolapse of iris or vitreous.

LANDOLT (Paris) objects to the suture even in case of

vitreous prolapse. A loose bandage, allowing the upper lid to move, is greatly to be preferred.

21. **Amyloid degeneration of conjunctiva and lids.** DEL MONTE (Naples) has found that the infiltration elements and plasma cells take part in the production of amyloid, as, particularly, do the mononuclear and polynuclear leucocytes. Red blood cells may also be concerned, after having been flooded into the tissues from the diseased vessels. Vascular changes are probably the first step in the degenerative process.

22. **Certain forms of strabismus.** ORLANDINI (Venice) discusses squints due to unilateral amblyopia, and to congenital amblyopia, as well as to intraocular disease, which are differentiated from the forms dependent on refraction error. The results of his examination and operative effects are reported in detail.

23. **Effect of perspective on form and size of images.** OVIO (Modena).

24. **Effect of perspective on acuteness of vision.** G. OVIO.

25. **Effect of perspective on reading.** G. OVIO studied the change in form of retinal images due to inclination in reading, and analyzes faulty positions in near work. Perspective plays an important part, influencing the size and position of images, and is greatly dependent on inclination in reading. Vision is equally affected in both eyes by inclination about a vertical axis (declination), and equally, forwards and backwards, in inclination about a horizontal axis. In reading letters of the alphabet, the disturbance of perspective is greater, with like degrees of inclination, after declination than after inclination. These inclinations are modified by fixation angles, which, in binocular vision, may not be equal, so that there is a difference of perspective in the images received by the two eyes. This is important for the hygiene of the eye which in reading requires large type, short lines, small size of volume, and medium distance both vertically and horizontally.

26. **Treatment of incipient senile cataract by subconjunctival injections of potassium iodide.** VERDERAU uses 2% solutions, injecting about 20 minims with 1% acoin. Improvement was noted in 26% of the cases.

27. **Ophthalmia medicamentosa sive therapeutica.** GUT-

MANN (Berlin) sounds a note of warning against meddlesome treatment, prolonged instillation of drops, or application of astringents, irritating salves, and so on. Many cases clear up when the treatment is stopped, particularly if the source of irritation is removed by correcting errors of refraction and muscular imbalance. Silver nitrate should not be used indefinitely; hot compresses are of use in keratitis, particularly scrofulous forms, in acute conditions only. In sac affections probes should be passed as seldom as possible. Atropin with cocain may cause infiltrations which may simulate purulent mycotic deposits. Cauterization then does harm. Subconjunctival injections of sodium chloride are less objectionable than those of mercury salts, particularly, the bichloride.

DUFOUR (Lausanne) supports Gutmann's views, particularly as to cocain.

28. **Etiology of pterygium.** ORTIN considers pterygium the result of a chronic irritation or disease of the conjunctiva.

29. **Toxic conjunctivitis of animal origin.** COSMETTATOS (Athens) describes a form of ophthalmia very similar to Parinaud's, with fleshy excrescences of the conjunctiva of the upper lid, and swelling of pre-auricular and submaxillary glands. There are no yellowish granulations however. The condition lasts several days, is attended with fever, and is probably due to the poison of an insect which has been injected under the skin by a sting.

30. **Extraction of senile cataract in the capsule.** ELSCHNIG (Prag) has tried the Indian method, or Smith's modification of Pagenstecher's operation, which differs from the latter only in the possibility of performing it without iridectomy. Of 34 simple expressions, there was vitreous prolapse in 8, iris prolapse in 12 (40%). Of 71 simple extractions, there was only 1 prolapse of vitreous, and 5.6% of iris prolapse, showing the great superiority of the latter method. Even in combined expression (29 cases) there was over 16% of vitreous prolapse, with otherwise good results finally and loss of sight in one case only, from glaucoma. Persistent irritation, corneal erosions, and marginal infiltrates are common. Expression should always be combined with iridectomy, and if the lens does not present readily, the capsule

should be opened. Loss of vitreous is due to the lens being held in the patellary space. Cataracta fusca is least suitable to expression in the capsule. The study, by A. Knapp, of 104 cases in Smith's clinic, agreed with the results of Elschnig, and shows slight loss of vitreous in 10, copious loss in 3, subchoroidal hemorrhage in 1, suppuration in 2, prolapse or incarceration of iris in 17 cases.

Discussion: GALLENGA (Parma) thinks extraction in the capsule should be used only for cataracta cystica, and in other cases permitted only if vitreous presents before capsulotomy.

AXENFELD (Freiburg) objects to extraction in the capsule as unsuitable for European conditions and thinks it should be abandoned as soon as possible.

HOWE (Buffalo) also condemns the method.

FALCHI (Modena) thinks extraction in the capsule the ideal operation, but, in its present state, suitable only for Morgagnian cataract, where it is very useful.

ELSCHNIG, in closing, confirms Axenfeld's views of the unsuitability of expression as a routine method.

(*To be continued.*)

REPORT OF THE PROCEEDINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. C. DEVEREUX MARSHALL.

An ordinary meeting of the Society was held at the Medical Society's Rooms, Chandos Street, on Thursday, June 10th, Mr. W. H. Jessop in the chair.

Mr. Freeland Fergus showed a case operated upon for paralytic ptosis by his method of resection. Mr. Arnold Lawson exhibited a case of double iridectomy for congenital glaucoma by the method of Lagrange, and Mr. Herbert Fisher, an unusual form of cataracts, bilateral, in a girl. Mr. Jessop brought forward a case of bilateral ptosis with complete ophthalmoplegia externa. Mr. Bergin showed a case with a unilateral swelling, and Mr. Blair one of pemphigus of the conjunctiva; while Mr. Herbert Bell exhibited a case of unusual elastic adhesion between iris and cornea. The latter was discussed by Dr. Weeks (of New York), Mr. Brooksbank James, Mr. Treacher Collins, Mr. Odillo Maher, and Mr. F. R. Cross.

Mr. P. H. Adams read two papers, the first on **A family with congenital displacement of lenses.** The series consisted of a mother and 7 out of her 9 children who suffered from this condition, but no other instance could be found in the family. In three of the cases the lenses were displaced downwards, instead of the more usual displacement upwards, while in the eldest boy the dislocation was complete, thus leaving the pupil clear of the lens. The two members of the family who were unaffected had perfectly normal eyes. The second paper was on **A family with congenital opacities of lenses.** In this family the great-grandfathers, grandmother,

father, and four of his children (out of five), as well as the father's sister, showed the peculiar opacities of the lens known as stellar cataract. Posteriorly there were from 3 to 6 primary rays, and numerous secondary ones, while some of them showed Y-shaped markings on the anterior surface as well. The opacities slowly increased throughout life, especially in the front of the lens, while the posterior markings were broader and less defined.

Messrs. A. Hill Griffith and A. W. Ormond read a paper entitled **A case of retinal disease with massive exudation and arterio-venous communication.** The patient was A. J., a female, æt. thirty-one. The left eye had been removed for secondary glaucoma by Dr. Hill Griffith ten years ago. The fundus of the remaining eye showed very tortuous and distended retinal vessels, with varicosity of several of them, and a red blurred optic disk. In the extreme periphery of the fundus, deep to the retinal vessels, was a whitish mass, the limits of which could not be seen. Issuing from the anterior border was a large vein, and running to it an artery somewhat similar in size and color. The changes in the fundus were probably all due to vascular disease. The interest in the case was furthered by the fact that the other eye of the patient was removed for secondary glaucoma simulating an intraocular growth, and on examination it proved to be a case of extensive subretinal exudation, with retinal detachment and an old organizing hemorrhage, but no growth. The patient's sister also lost an eye from a similar cause, and the same condition was found on pathological examination.

Dr. George Mackay reported a very unusual appearance—an epithelial filament simulating a threadworm in the anterior chamber of the eye of a little girl, aged seven. The child had always enjoyed good health, had no special illness, had never suffered from worms, and had not complained much about the eye. The filament had been first noticed when she was eight months old. No independent movement had ever been detected in it, but the parents were under the impression that it was slowly increasing in size. The eye was occasionally a little irritable, with a tendency to lachrymation and a trace of ciliary injection. The tension was normal, the media otherwise transparent, and the fundus healthy.

The pupil responded to light, though less mobile below than above. At the periphery of the iris, close to the anterior chamber angle, between the 5 and 8 o'clock meridians, a buffy-gray filament about a millimetre in diameter, emerged between the fibres of the iris, ascended in close relation to the posterior aspect of the cornea for three or four millimetres, then bent backwards towards the lower border of the pupil, and, crossing the vertical meridian of the anterior chamber, appeared to rest lightly in contact with the sphincter surface of the iris. Thereafter it bent downwards once more, following the plane of the iris nearly in the 7 o'clock meridian. The structure terminated in a slightly bulbous free extremity, which did not re-enter the iris, nor extend to the angle of the anterior chamber. The arched filament had thus a slight spiral twist from before backwards. The refraction of the eyes was about emmetropic. The vision of the right eye $\frac{6}{12}$ fairly, and the left, $\frac{6}{6}$. The child was put under chloroform, and through an incision made at the corneo-scleral junction, a little to the nasal side of the filament, the foreign body was successfully removed, though breaking into two pieces on account of its brittle structure. From the pathological examination made by Dr. Shannon of Edinburgh, and Mr. A. E. Shipley of Cambridge, no confirmation was obtained of the presence of a parasite. Microscopic sections showed that the filament was mainly composed of stratified epithelium, with a little loose connective tissue and some melanin pigment. Discussion was invited as to its probable origin, *e.g.*, in a congenital displacement of part of the epithelium from the anterior surface of the iris, or a capsulo-pupillary vestige.

Mr. W. H. H. Jessop gave a report of the International Committee on the **unification of the notation of visual acuity and of the meridians of astigmatism.** The Committee, after consideration, came to the following conclusions: (1) That the meridians of astigmatism should be measured and represented as the observer looked at the patient. (2) That the axes should be measured and represented in the lower semicircle of the trial frame. The numbering of the axes should start from the middle line of the face in each eye and proceed downwards and temporalwards. The zero would therefore lie at the nasal end of the semicircle and 180° at the

temporal end; 90° would be below and midway between these points. The Committee also agreed that for international test type, numbers should be used. Landolt's ring was also thought to be a suitable object. The unit of measure was an angle of 1 minute, and the figures were to be constructed on this unit. The types were to be constructed as regards size on arithmetical progression, and the standard distance at which they were to be seen should be 5 metres. The source of light was to be diffuse daylight, if possible, opposite the types, and not laterally.

An ordinary meeting of the Society was held at the Rooms of the Medical Society on Friday, July 9th, Mr. PRIESTLEY SMITH occupying the chair.

Dr. F. W. EDRIDGE GREEN showed a spectroscope for testing color perception. It is constructed like an ordinary spectroscope, except that there are a telescope and collimator both fixed, and in the focus of the eyepiece there are two shutters. These cover the whole of the spectrum, and by means of them the operator can isolate any portion of the spectrum, in order to show the size of the patch, which seems to be monochromatic to any person. He said that whereas it used to take him three days to examine a case by the old instrument, he could now examine one in half an hour, because attached to each shutter is a drum marked in wave lengths, graduated to the fourth degree, which was very fine. That enables the size of the patch to be determined without any calculation. The spectroscope could be used for seeing the exact termination of the spectrum, and how far the ends extended. Mr. N. BISHOP HARMON showed a chart illustrating some new cataract pedigrees. The cases illustrated the truth of Mr. Treacher Collins's contention that the cataract was a defect in the lens secretion which went to make the capsule. The patients had plaques on the back part, behind the nucleus, and including the posterior part of the lens. In one girl the cataract extended so as to include the nucleus, while in a further case it projected forward like a piece of coral through the lens, so that it seemed there was lens matter bulging through. At first he thought it was that, and that it had been needled, and afterwards got hard and sclerosed.

Mr. ARNOLD LAWSON and Mr. MACKENZIE DAVIDSON read

a paper entitled **A preliminary note on the treatment of eye diseases by radium.** Mr. LAWSON said the cases had been selected from the out-patient clinique of Moorfields Hospital, and were selected as instances of maladies which usually proved obstinate or only improved slowly under the ordinary forms of treatment, or else were of an active and virulent nature and so required drastic measures. Only 17 cases had so far been tried, but in every case the result of exposure to radium had been very encouraging. Most of the cases were corneal ulcerations, four were non-ulcerative lesions, and one was episcleritis. After one application of five minutes to a hypopyon there was distinct benefit. For instance, a man aged thirty-five, gave a history of injury to the right eye. There was deep ulceration, and a gray infiltrated area of 4*mm* diameter, with a large hypopyon; 29 milligrams of radium were applied for 5 minutes, and five days later the injection was subsiding, and the hypopyon had disappeared. Two days after a second application of three minutes the man was discharged from the hospital, the eye being not quite white but, for all practical purposes, well. Other cases also did very well from the treatment. In all cases the only other measures were boracic lotion and atropin where required. Some punctate erosions were also similarly treated, and gave equally encouraging results, as also did cases of old-standing trachomatous pannus, except in one case, in which there was increased vascularity after the treatment. The dosage of radium was an important point, and the small dose of 5 milligrams seemed to act as quickly as did a much larger quantity. In one case the pain after the application endured three days, but in the others the longest time was 20 hours. Dr. Mackenzie Davidson explained that the paper was a tentative one. The radium was applied in sealed glass tubes, the glass cutting off the Alpha rays, so that the Beta and Gamma rays only passed through to the lesion. Mr. ERNEST CLARKE and Mr. HEWKLEY discussed the paper.

The Chairman then, in a graceful tribute, presented to Mr. EDWARD NETTLESHIP the Nettleship medal for his work in ophthalmology, and Mr. NETTLESHIP replied, paying a high compliment to the many coadjutors who had helped him to complete the pedigrees on which he had been working.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. JOHN R. SHANNON, Secretary.

MEETING OF MONDAY, MAY 17, 1909. DR. W. E. LAMBERT, PRESIDENT, IN THE CHAIR.

Present, thirty-five members.

Dr. E. S. Thomson, for Dr. David Webster, presented a case of **sarcoma of the orbit, removed by Kroenlein's operation.**

The tumor was found at the upper and inner side of the orbit. It was one and one-half inches long, flat, and lying close to the periosteum. The exophthalmos remained a month, and then subsided. Vision is $\frac{20}{20}$. The growth was a small, round-celled sarcoma.

Dr. H. W. Wootton presented, 1st, a case of **needling of dense after-cataract through the sclera (scleronyxis).** The needle was inserted *7mm* behind the limbus, the incision being made subconjunctivally. The membrane was very easily cut, and there was no reaction. 2d, a case of **thrombosis of the superior retinal vein.** The hemorrhages are limited to the upper field. The patient was unaware that there was any trouble with the eye, and came merely for glasses. Vision was $\frac{20}{50}$. The external coats of the vessels did not seem to be involved. The radial pulse is normal. Heart and kidneys likewise normal.

Dr. John Leshure presented a case of **foreign body in the crystalline lens.** The boy's eye had been injured two years previously by the explosion of a percussion cap, a bit of which is lodged in the lens. There are no iritic adhesions, and there is no irritation. He asked what was best to be done.

Dr. E. B. Coburn presented a boy who came to Cornell

on the fifth of May, with trachoma and pannus. Expression was done. Examining the lower lid, he discovered that in addition to the normal puncta there was a secondary groove communicating with the lachrymal sacs.

Dr. MARTIN COHEN presented a case of **tuberculosis of the sclera** in a colored man thirty-five years old.

The family history and previous history of the patient negative. Right eye became inflamed six months ago, and very painful. Vision gradually became impaired. Cough has been present for several weeks. Appetite good; no night sweats or loss of weight. No evidence of any abnormal condition, except in the eye. Vision of the left eye $\frac{20}{10}$; one dioptry hypermetropia. Vision, right eye, counts fingers at two feet. Field concentrically contracted. Tension plus. Anterior chamber clear and of normal depth. Pupil regular at the nasal half; iris markings indistinct; extending over nearly the entire ciliary region, and also over the lower and inner quadrant of the eyeball, are nodular masses involving the sclera. The entire bulbar conjunctiva is injected. Vitreous is cloudy, owing to choroidal exudate. The Wasserman reaction was negative, also anti-syphilitic treatment carried on for four months. Von Pirquet reaction was positive. Case appears to be one of scleritis of tuberculous origin. Patient is at present receiving tuberculin injections with, so far, very slight improvement.

Dr. JOHN R. SHANNON reported a case of **severe poisoning by homatropin.**

The patient was a young lady who came for examination of the refraction four weeks ago. Two years previously the eyes had been examined and glasses prescribed for mixed astigmatism. The records showed that at that time homatropin was instilled, with no ill effect. On this occasion, however, the patient showed disinclination to having the drug used, expressing a fear that a mistake might be made in putting it up, and that she might suffer harm thereby. She was reassured, however, and given a prescription for one gramme of two per cent. solution of hydrobromate of homatropin, two drops to be put in each eye every five minutes for half an hour, beginning one hour before the time of her appointment. Shortly before this time, Dr. Shannon was told over

the telephone that the patient had collapsed after three instillations of the drug. She was seen by a physician residing in the house, who administered restorative measures. Subsequently, she was seen by her family physician, who reported that she was totally unconscious and restless. Temperature 100°; pulse 112; respiration 24; pupils widely dilated; face very red; mouth and throat parched. The coma was gradually succeeded by delirium, but the circulation was still very poor six hours later. Conditions gradually improved from this time on, although for several days the patient complained of extreme weakness, and the pupils remained dilated for one week. The solution was examined at the Lederle Laboratories, and found to be homatropin. The literature on the subject was quoted.

Dr. ALEXANDER DUANE made a report of investigations on the **mydriatic action of homatropin.**

He reported the results of experiments in over five hundred cases. Using the very delicate test described by Uhthoff, he found that it usually took about twenty-five minutes for the light reaction to be completely abolished, although in some it disappeared in less than twenty minutes, and in some a faint reaction persisted for forty minutes, or even an hour. It lasted longest in persons from sixteen to twenty-five, while in those over forty, and especially over forty-five, it disappeared quickly. Quite a contrary result, however, is obtained if we measure the mydriatic action of homatropin by the amount of dilatation it causes—the smallest dilatation occurring at the age when the reaction disappears the quickest, and *vice versa*. In many cases, but not all, the pupil continues to dilate a little (0.5*mm*) after all reaction has ceased. The pupil generally begins to dilate in twelve to fourteen minutes after the first instillation, dilatation then often occurring quite suddenly. The accommodation is usually affected before the pupil begins to dilate—this being so at all ages. The addition of cocaine enhances the rapidity, and, to some extent, the amount, of dilatation, but not the amount of cycloplegia. When cocain was added to the homatropin, there seeemd to be a tendency for the pupil to dilate quicker and the reaction to cease sooner in the left eye than in the right. Answering Dr. Coburn, Dr. DUANE said there was

absolutely no relation between the amount or the rapidity of the mydriasis and the completeness of the cycloplegia. The pupil may be small and yet there may be no accommodation left, while a pupil of 8*mm* may co-exist with several dioptries of accommodation. In particular, the cessation of pupillary reaction is no indication that the cycloplegia is anywhere near complete.

Dr. E. GRUENING reported a case of **sub-hyaloid hemorrhage involving a section of the optic papilla.**

In the examination of thirteen of these cases, the hemorrhage had been found between the nerve fibres and the lamina limitans interna. Dr. Gruening's is the first case on record where the papilla was encroached upon. Dr. GRUENING also presented a case of **cow-horn injury of the eye, with a specimen.** This was the first case of the kind he had seen in New York City in a practice of forty years.

Dr. J. H. CLAIBORNE reported a case of **vacuole of the lens** and showed a drawing of the condition. The patient was first seen twelve months ago. Patient said that she had been struck several times in the left eye. The sclera was red, the cornea cloudy, posterior surface of the cornea studded with small dots, so that the iris could scarcely be seen. There was no fundus reflex. Tension was slightly raised. Vision: light perception. Diagnosis of diffuse choroiditis was made. Treatment: large doses of iodide of potash and atropin. Dr. Claiborne found that, by following the suggestion of Dr. Wilmer of Washington, if a small quantity of powdered nutmeg be given with the iodide, the patient was able to take 175 grains at a dose, instead of 75, her previous limit. After the third month, inunctions of mercury were begun. Gradually the conditions improved and it was discovered that the lens had been dislocated. When the fundus could be seen, it was found that the optic nerve was normal and there was a large patch of choroidal atrophy at the inner side of the fundus. At the end of the eighth month the lens became very clear. There were alternating radiations of clearness and opacity and between and inwards was what Dr. Claiborne described as a vacuole of the lens. In fact, there were two of these so-called vacuoles. Dr. Claiborne thought that these vacuoles probably contained

Morgagnian fluid. Dr. Claiborne quoted the literature on the subject.

Dr. CLAIBORNE also presented a **pocket ophthalmoscope,** which he demonstrated.

Dr. ALEXANDER DUANE gave a **demonstration of a test card for determining the range of accommodation.**

Dr. E. B. COBURN reported a case of **purulent uveitis,** which seemed to him to be due to an endogenous infection.

In the discussion of Dr. Webster's case (small-celled sarcoma), Dr. H. W. WOOTTON questioned whether the orbit should not have been exenterated. Dr. W. E. LAMBERT expressed similar views. Dr. THOMSON said that the decision in such a case is difficult. The vision was perfect, and experience shows that many such growths are atypical, and may never recur.

Dr. J. H. CLAIBORNE said that, as Dr. Bull had pointed out, those cases which are disturbed are the more apt to have recurrences. He was glad to find Dr. Thomson sustaining the clinical view in such a case in spite of the microscopic findings.

In the discussion of Dr. Wootton's case of scleronyxis, Dr. W. E. LAMBERT thought that that proceeding was dangerous, and preferred the De Wecker scissors.

In the discussion of Dr. Leshure's case, Dr. W. E. LAMBERT suggested an X-ray picture should be taken, subsequently iridectomy done, with the removal of the foreign body. Dr. E. S. THOMSON was of the opinion that the foreign body would best be removed by taking hold of the membrane which contained it, rather than the foreign body itself.

Dr. Coburn's case was discussed by Dr. J. H. CLAIBORNE.

In the discussion of Dr. Cohen's case, Dr. H. W. WOOTTON said that this did not correspond to the cases called tuberculosis of the sclera. He had seen a case at the Manhattan Hospital of deep scleral inflammation. The mother died of tuberculosis. Inoculations from the growth killed a guinea-pig and gave tuberculous iritis to a rabbit. The patient died of meningitis. The eye showed tuberculosis of the ciliary body, which had perforated the sclera. Two years ago, when he had looked the matter up, he had found but one

case of tuberculosis of the sclera—Mueller's. All others resembling it were cases of tuberculosis of the uveal tract. Dr. E. B. COBURN thought a growth should be examined for the spirochæta pallida.

In the discussion of Dr. Shannon's case, Dr. E. GRUENING said that he always instilled the homatropin himself, using a four per cent. solution of Merck's homatropin. He had never seen such a case in the thousands that he had examined under homatropin. He thought that the drug must have been atropin.

Dr. ALEXANDER DUANE quoted a case reported by Dr. Marple and said that he considered such cases merely idiosyncrasies. The case was also discussed by Drs. COBURN, CLAIBORNE, KRUG, LAMBERT, and BOWLES.

In the discussion of Dr. Claiborne's pocket ophthalmoscope, Dr. GRAEF stated that he had used an instrument almost precisely similar for several years.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE FIRST AND SECOND QUARTERS OF THE YEAR 1908.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the assistance of Prof. A. ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALÉN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA, Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTELSTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and others.

Translated by Dr. PERCY FRIDENBERG, New York.

Sections VIII.–XII. Reviewed by Prof. SCHWEIGGER, BERLIN.

VIII.—LIDS.

126. MORAX and CARLOTTI. **Palpebral sporotrichosis.** *Ann. d'ocul.*, cxxxix., p. 418.

127. TERTSCH, R. **Cases of vaccine inoculation of the eye.** *Wiener klin. Wochensch.*, 1908, No. 2.

128. HOFFMANN, R. **Degeneration of lashes, eyebrows, and nails in thyroid disease.** *Arch. f. Dermatol. u. Typh.*, lxxxix., No. 3.

129. BOTTINI, F. **Electrolytic depilation in partial trichiasis.** *Ann. di Ottalm.*, xxxvii., Nos. 1 and 2.

130. MARONGUI, L., and MARCHI, F. **Streatfield section (modified) and transplantation of labial mucosa in entropion.** *Ann. di Ottalm., ibid.*, Nos. 3 and 5.

131. NICATI, W. **My operation for entropion of upper lid.** *Ann. d'ocul.*, cxxxix., p. 344.

132. VOSSIUS. **Pitchfork injury of superciliary ridge and optic nerve.** *Deutsch. med. Wochenschr.*, 1908, No. 38.

133. PFALZ. **Early transplantation of flaps in burns of lids.** *Ibid.*, No. 19.

134. ORLOFF, K. **Alveolar echinococcus of upper lid.** *Ann. d'ocul.*, cxxxix., p. 117.

135. MARKBREITER, J. **Lid changes due to improper prothesis.** *Klin. Monatsbl. f. Augenheilkunde*, xlvii., No. 1, p. 637.

136. STEINER, L. **Contribution to surgery of the lids.** *Ann. d'ocul.*, cxxxix., p. 112.
137. PICK. **On canthoplasty.** *Deutsche med. Wochenschr.*, 1908, No. 13.
138. FALCHI, F. **Blepharoplasty in central coloboma of the lid.** *Arch. f. Augenheilkunde*, lix., p. 226.
139. NANCE, W. **Congenital coloboma of lid.** *Journ. of Ophth. and Oto-Laryngol.*, January, 1908.
140. MORAX, V. **Palpebral autoplasty with pedunculated flaps (Snydacker).** *Ann. d'ocul.*, cxxxix., No. 14.
141. MIRTO, G. **Psychogenic nature of blepharospasm.** *Clin. oculist.*, 1908, pp. 5–6.
142. VALUDE, E. **Blepharospasm and alcohol injections.** *Ann. d'ocul.*, cxxxix., p. 241.
143. SHOEMAKER, W. **Motais operation for ptosis.** *Ann. of Ophth.*, October, 1907.
144. AHLMANN, B. **Treatment of cancer with radium.** *Westn. Ophth.*, 1908, No. 2.
145. COSMETTATOS, G. **Granuloma of lid margin.** *Arch. f. Augenheilkunde*, lix., p. 413.
146. MORAX, V., and BEAL, R. **Pigmented nævus of brow. Plastic operation.** *Ann. d'ocul.*, cxxxix., p. 34.
147. CALDERARO. **Dermoid of lower lid.** *Clin. ocul.*, 1908, No. 1.
148. NICOLAI. **Case of true keloid of upper lid.** *Ned. Tydschr. v. Geneesk.*, 2, 1.
149. REIS, W. **Ankyloblepharon filiforme adnatum.** *Arch. f. Augenheilkunde*, lvii., No. 4, p. 283.
150. WINTERSTEINER. **Case of ankyloblepharon adnatum.** *Ibid.*, lix., p. 196.
151. WEILL, G. **Case of ankyloblepharon adnatum.** *Ibid.*, p. 278.

MORAX and CARLOTTI (126, **Palpebral sporotrichosis**) saw, in a man of seventy, ulceration of the lid margin with subcutaneous abscesses, lymphangitis of the cheek, and swelling of the preauricular and submaxillary glands. Treatment with iodides resulted in a cure.

TERTSCH (127, **Cases of vaccine inoculation of the eye**) observed inoculation ulcers of lids and conjunctiva in a child of ten after removal of a foreign body by vaccinated relatives. Ulcer of the cornea also developed and healed in the course of time.

HOFFMANN (128, **Degeneration of lashes, eyebrows, and nails in thyroid disease**) noted this condition in a woman of twenty-seven, and in ancestors and descendants. The same condition is found in infantile myxœdema. Signs of hyperthyroidism were marked.

Bottini (129, **Electrolytic depilation in partial trichiasis**) reports 64 cases of partial trichiasis treated with good functional and cosmetic results.

Nicati (131, **My operation for entropion of upper lid**) everts the upper lid, and dissects off the shrunken conjunctiva from the tarsus which is then cut through and sutured after Panas.

Pfalz (133, **Early transplantation of flaps in burns of lids**) advises this procedure in all cases where cicatrices are to be feared. Thiersch flaps are used which are anchored in the fornix fold. This should be done from four to six days after the burn.

Orloff (134, **Alveolar echinococcus of upper lid**) describes a tumor of the upper lid resembling a fibroma the size of an almond. It was near the margin of the superior tarsus and adherent to the fibres of the orbicularis muscle.

Markbreiter (135, **Lid changes due to improper prothesis**) describes a notch 5*mm* deep in the lower lid with hard margin and excoriated surface. Microscopic examination showed adenoid tissue.

Pick (137, **On canthoplasty**) makes an incision which after cutting through the tarsus divides in the conjunctiva making a pedicle which is sewed into the skin wound so as to avoid traction on the conjunctiva and cutting out of the sutures.

Falchi (138, **Blepharoplasty in central coloboma of the lid**) splits the outer commissure horizontally and then makes two oblique cuts in the direction of the coloboma, one at the angle, and one at the temporal prolongation of the enlarged lid fissure. This flap is then loosened and slid nasally. The defect is covered by Thiersch flaps.

Morax (140, **Palpebral autoplasty with pedunculated flaps (Snydacker)**) describes a modification of Snydacker's procedure of skin flaps from the neck. The flap is only partially separated and sewed to the margin of the lid defect at the first operation. Several weeks later the other end is brought in place and fastened.

Mirto (141, **Psychogenic nature of blepharospasm**) reports two cases, one treated by section of the facial. After apparent cure there was a relapse. In the second case, injections of alcohol were followed by neurectomy of the ophthalmic branch of the trigeminal, without effect.

VALUDE (142, **Blepharospasm and alcohol injections**) reports 3 cases, two of which were cured, but in one there was keratitis from lagophthalmus. Tic douloureux and facial hemispasm were noted in two cases.

SHOEMAKER (143, **Motais operation for ptosis**) describes the transplantation of the middle third of the tendon of the superior rectus into the upper lid between tarsus and skin. An incision through the skin of the lid facilitates this step. A small, thin tendon is a contraindication.

AHLMANN (144, **Treatment of cancer with radium**) reports 7 cases. In one case there was a recurrence after eight months, in 2 after a year, in one after two years. In one case there was no recurrence after two years. The other 3 cases could not be followed up.

COSMETTATOS (145, **Granuloma of lid margin**) notes that these growths are usually benign, barley corn to pea size, round, raspberry red, and pedunculated. Polypoid granulomata may be due to trauma and may become malignant as in his case, aged thirty-five, which grew rapidly.

MORAX and BEAL (146, **Pigmented nævus of brow**) removed a new growth and covered the defect by Snydacker's procedure. A dermoid of the conjunctiva was also present.

CALDERARO'S (147, **Dermoid of lower lid**) dermoid was the size of a pigeon's egg. It contained giant cells and was due to epiblastic inclusions.

NICOLAI (148, **Case of true keloid of upper lid**) reports the case of a youth of seventeen with growths in the upper lids extending like cords up to the brow. Microscopic examination showed that they were circoid neuromata.

REIS (149, **Ankyloblepharon filiforme adnatum**) found this condition in a new-born child at the middle of the lid fissure. It could be stretched to a length of 5*mm* and consisted of elastic tissue and vessels. It was not due to developmental anomaly and could be explained by injury to the lid by the finger nails of the embryo and subsequent adhesions of the lid margin.

WINTERSTEINER (150, **Case of ankyloblepharon adnatum**) noted a similar growth in post-mortem examination of a premature birth. The filiform band grew from the intermar-

ginal space. The fibres of the orbicularis were affected by the cicatricial contraction of this cord.

WEILL (151, **Case of ankyloblepharon adnatum**) describes a cord-like band between the middle and outer third of the lid margin. It was ½*cm* long and drew slightly on the opened lids, and became shrunken after being cut.

IX.—LACHRYMAL APPARATUS.

152. MAYOU. **Lachrymal abscess in the new-born.** *Royal London Ophth. Hosp. Reports*, January, 1908.

153. ELLIOTT, R. **Three hundred cases of extirpation of lachrymal sac.** *Ophth. Rev.*, February, 1908.

154. GROENOUW. **Extirpation of orbital lachrymal gland.** *Centralbl. f. prakt. Augenheilkunde*, xxxii., p. 100.

155. CONSTANTIN. **Traumatic prolapse of lachrymal gland. Reposition.** *Arch. d'opht.*, xxviii., p. 243.

156. KUELBS. **Mikulicz's disease.** *Mitt. a. d. Grenzgeb. d. Med.*, xviii., No. 5, p. 754.

157. AUBARET. **Valvular insufficiency of lachrymal canal.** *Arch. d'opht.*, xxviii., p. 211.

158. AUBARET. **Valvular folds of canaliculi and duct.** *Ibid.*, p. 347.

159. LOTIN, A. **Electrolysis in fistula of sac.** *Arch. f. Augenheilkunde*, lx., p. 197.

160. KRAUSS, W. **Technique of extirpation of sac.** *Arch. f. Augh.*, lix., 351.

161. SCIMENI, E. **Simple method of extirpation of sac.** *Clin. oculistica*, March, 1908.

162. KOSTER, W. **Permanent drainage of lachrymal passages.** *Graefe's Arch. f. Ophth.*, lxvii., No. 2, p. 377.

163. BERNHARDT, H. **Lachrymal atresia in the new-born.** *In.-Diss.*, Rostock, 1907.

MAYOU (152, **Lachrymal abscess in the new-born**) contributes an excellent article which should have been headed "The Anatomy and Development of the Fœtal Lachrymal Apparatus." Lachrymal abscess appears during the first few weeks of life, and is primarily due to obstruction of the lachrymal duct with a secondary infection with the gonococcus or other organism. Mayou has found the gonococcus, the pneumococcus, and the staphylococcus and the Morax Axenfeld bacillus. These were never present in pure culture. Mayou has examined fœtuses at the tenth week, and the third, fourth, and fifth month of gestation; also five at term.

They were hardened in formol, and cut in serial section. When necessary decalcification was carried out with sulphurous acid. The results obtained are as follows: The *canaliculi* are formed before the tenth week by an infolding of the lid margin. At the tenth week they consist of solid columns of cells. At the twelfth week a lumen is present lined by three or four layers of squamous epithelium.

The *sac and duct* are developed in the facial cleft as a folding in of epiblast. The lower end of the duct is first differentiated, but it has no lumen till the fourth month. Bone is deposited around the lower end first. The lachrymal sac never loses its primary lumen. It is the last part of the facial cleft to close.

Anatomy at Birth. Only a small part of the inferior meatus, close to the septum is patent. The turbinals are packed closely together, the inferior resting on the floor of the nose. The interstices of the bones, the duct, the sac, and the canaliculi are filled up with debris. If the canaliculi be injected with any force extravasation always takes place in the situation where an abscess is usually found. In the upper part of the groove in which the lachrymal duct lies above the internal palpebral ligament the injection passed directly round the sac. In the lower part, between the internal palpebral ligament and the entrance of the duct into the bony canal, the extravasation took place forwards into the lower lid. The reason is obvious. The fascia covering the internal rectus muscle passing forwards gives off a slip which blends with the periosteum of the lower lip of the lachrymal groove, whilst it passes forward as the internal check ligament to be attached to the eyelids and orbital margin below while next to the muscle it blends with the capsule of Tenon. This fascia, therefore, bounds a triangular space with its apex directed backwards, the inner wall being made up of the periosteum of the lachrymal bone and the attachment to it of the slip of the internal check ligament, the outer wall being formed by the internal check ligament, while the anterior wall is formed by the skin and orbicularis muscle, etc., and is crossed by the internal palpebral ligament. The lachrymal sac is situated on the inner wall of the space supported on its inner and posterior surfaces by bone so that when infection

takes place an abscess forms in the space described, it cannot pass backwards into the orbit on account of the check ligament and must come forwards, and point above or below the palpebral ligament.

Mayou concludes that (1) Throughout development the lower end of the lachrymal duct is small, and remains so at birth, being partially occluded by the pressure of the inferior turbinate bone. (2) That at birth the sac and duct are filled with epithelial debris. (3) That at birth the duct is either not patent or not fully so. It is a clinical fact that one passage of a probe will clear the duct, and prevent the formation of an abscess.

The paper is enriched by four excellent plates.

T. Harrison Butler.

Elliott (153, **Three hundred cases of extirpation of lachrymal sac**). The method adopted by Elliott in extirpating the lachrymal sac is hardly as refined as that described by Meller in his new book detailing the operative methods adopted in Fuchs's Clinic at Vienna. Elliott's incision from 15 to 27*mm* long, and averaging 20*mm*, seems an unnecessarily long one. It only commences at the lower border of the internal palpebral ligament which is not divided. It must be very difficult with such an incision to remove the dome of the sac, one third of which lies above the ligament. Three assistants are mentioned, whereas, under local anæsthesia, which properly carried out admits of an almost painless operation, the sac can be removed often bloodlessly and generally single handed. Then when the sac has been dissected out a red-hot spindle-shaped cautery is thrust down the duct, a needless and quite unsurgical action, and one that necessitates a general anæsthetic. The dome of the wound is to be freely cauterized when necessary with a red-hot ball-shaped cautery. The actual cautery is even used to check hemorrhage. We ask—"why these mediæval measures?" Why not neatly dissect out the sac and sew up the wound? It practically always heals by first intention and the case is cured.

The indications for the operation are such as commend themselves to all modern surgeons, but we would go further and say have no probes at all. If simple syringing will not

cure the case remove the sac, which seems to fill a very small space in the economy of nature. T. HARRISON BUTLER.

GROENOUW (154, **Extirpation of orbital lachrymal gland**) reports a cure by this procedure for obstinate epiphora following extirpation of the sac, after removal of the palpebral portion had been performed without effect.

CONSTANTIN (155, **Traumatic prolapse of lachrymal gland**) noted this accident in a child of eighteen months. Reposition in narcosis was permanent.

KUELBS (156, **Mikulicz's disease**) saw a young man with bilateral swelling of parotid, submaxillary, and lachrymal gland of fourteen days' standing. Hard palate and lymph glands normal. Blood, and so on, normal. Large doses of iodides and inunctions brought about a gradual cure. Microscopic examination showed chronic inflammation with round-cell infiltration. No changes of the lymph apparatus; no tuberculosis, malignancy, or pseudoleukemia. Etiology was not determined.

AUBARET (157, **Valvular insufficiency of lachrymal canal**) distinguishes between mechanical or physiological insufficiency dependent on severe pain which may be intermittent, and a pathological form due to tissue changes in the canals or sac. Valvular anomalies are frequent.

AUBARET (158, **Valvular folds of canaliculi and duct**) often finds insufficiency of Hasner's fold so that air is admitted from the nasal passages. The valves above the sac are rarely defective.

LOTIN (159, **Electrolysis in fistula of sac**) finds this method more rapid and effectual than probing or syringing. Suppuration disappears rapidly, even after one sitting. In fistula of the sac the naso-lachrymal canal must be destroyed electrolytically by sound.

KRAUSS (160, **Technique of extirpation of the sac**) avoids injuring the ligamentum palpebrale internum and the periosteum behind the sac. Curved skin incision, blunt dissection, avoiding the vessels. Acoin-adrenalin-cocain anæsthesia. No assistants are needed.

SCIMENI (161, **Simple method of extirpation of sac**) injects pyoktanin 1%, 1*cc* during local anæsthesia. The extirpation is indicated in lachrymal fistula, chronic suppurative dacryo-

cystitis, blennorrhœa with dilatation, mucocele, old stenoses complicated with corneal ulcer, and preparatory to operations on the eyeball, as well as in obstinate cases.

BERNHARDT (163, **Lachrymal atresia in the new-born**) believes that this condition explains the cases usually described as blennorrhœa of the sac. Expression of the sac is usually sufficient. Probing may be necessary. Inflammation of the sac is rare in children, and may be due to periostitis and accessory sinus disease.

X.—ORBIT AND ACCESSORY CAVITIES.

164. REBER, W. **Orbital abscess secondary to nasal disease.** *Am. Journ. of Ophth.*, February, 1908.

165. RISLEY, S. **Binocular exophthalmus. Recovery.** *Ophth. Record*, April, 1908.

166. EVANS, J. H. **Topographical anatomy of the sinuses and orbit.** *Ophthalmoscope*, April, 1908.

167. EVANS, J. J. **Ocular and orbital complications of accessory sinus disease.** *Ibid.*, April, 1908.

168. ST. CLAIR THOMSON. **Frequency of orbital manifestations of nasal sinusitis.** *Ibid.*

169. KATZ, R. **Treatment of tenonitis.** *Westn. Ophth.*, 1908, No. 2.

170. RUS, M. **Orbital phlegmon due to dacryocystitis. Cure.** *Wiener med. Wochenschr.*, 1908, No. 4.

171. HOFFMANN, R. **Relation of orbital phlegmon to sinusitis.** *Deutsch. Laryng. Gesellsch.*, Dresden, 1907.

172. BIRCH-HIRSCHFELD, A. **Relation of orbital inflammations to accessory sinus disease.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 1.

173. KUHNT, H. **Relation of nasal and ocular disease.** *Deutsche med. Wochenschr.*, 1908, No. 37.

174. ONODI, A. **Relation of nasal and ocular disease.** *Berl. klin. Wochenschr.*, 1908, No. 18, 19.

175. BRUECKNER, A. **Relation of eye and nose in disease.** *Arch. f. Augenheilkunde*, lviii., No. 4, p. 316.

176. CHIARI and MARSCHIK. **Disease of orbit and accessory sinuses.** *Med. Klinik*, 1908, p. 576.

177. NEWOLINA, A. **Shot wound of orbit.** *Deutschmann's Beitr.*, 1908, No. 70, p. 594.

178. WILLIAMS, E. **Bullet wounds of orbit.** *Ophth. Record*, June, 1908.

179. PECHIN and DESCHAMPS. **Orbital injury and alternate hemiplegia.** *Soc. d'opht. de Paris*, March 10, 1908.

180. DUBOIS DE LAVIGERIE. **Spontaneous luxation of globe.** *Ibid.*, January 14.

181. NATANSON, L. **Microphthalmus and anophthalmus congenitus with orbital cysts.** *Arch. f. Ophth.*, lxvii., No. 2, p. 185.

182. BASSO, D. **Tumors of the orbit. Kroenlein operation.** *Arch. f. Augenheilkunde*, lix., p. 281.

183. LEWIT, B. **Operative removal of orbital exostosis.** *Wratsch. Gaz.*, 1908, No. 18.

184. SCHMIDT-RIMPLER. **Orbital osteoma due to periostitis.** *Muench. med. Wochenschr.*, 1908, No. 12.

185. FEHR. **Lymphangioma cavernosum orbitæ.** *Centralbl. f. prakt. Augenheilkunde*, xxxii., p. 134.

186. KOSTER, W. **Spontaneous cure of sarcoma (?) of orbit.** *Zeitschr. f. Augenheilkunde*, xix., p. 28.

187. WAGENMANN, A. **Recurrence of melanosarcoma eleven years after enucleation.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 140.

188. WATANABE. **Intravascular endothelioma of orbit.** *Arch. f. Augenheilkunde*, lvii., No. 4, p. 269.

189. LODATO, G. **Cavernous angioma of orbit.** *Arch. di Ottalm.*, xv., No. 7, 1908.

190. CANTONNET and OFFRET. **Cavernous angioma of orbit. Optic atrophy.** *Soc. d'opht. de Paris*, April, 1908, p. 67.

191. EWETZKY, F. **Significance of orbital teratoma.** *Westn. Ophth.*, 1908, No. 3.

192. SCHWALBACH. **Inflammatory exophthalmus.** *Deutsche med. Wochenschr.*, 1908, 22, p. 910.

193. BERTRAM. **Congenital bilateral exophthalmus.** *Arch. f. Augenheilkunde*, lix., p. 378.

194. BEAUVOIS, A. **Exophthalmus in maxillary sinusitis of the new-born.** *Recueil d'opht.*, xxx., p. 54.

195. NICOLAI, W. **Case of traumatic enophthalmus.** *Arch. f. Augenheilkunde*, lx., p. 261.

196. TEICH, M. **Traumatic enophthalmus.** *Ibid.*, p. 266.

197. VAN DUYSE. **Traumatic enophthalmus.** *Arch. d'opht.*, xxviii., No. 1.

198. TOBIAS. **Treatment of exophthalmic goitre.** *Zeitschr. f. Diaet. u. phys. Ther.*, xii., 2.

199. OHLEMANN, M. **Exophthalmic goitre.** *Muench. med. Wochenschr.*, 1908, p. 36, 1883.

200. PREYSING, H. **Perforation of orbit in sinusitis due to scarlet-fever.** *Deutsche Zeitschr. f. Chir.*, xciv., Nos. 3 and 4.

201. RICK, O. **Osteoma of frontal sinus. Kuester's operation.** *Inaug.-Diss.*, Marburg, 1907.

202. STERN. **Congenital anomaly of ethmoid labyrinth.** *Deutsche med. Wochenschr.*, 1908, No. 22.

203. TRAUTMANN, G. **Sphenoid suppuration with intracranial and orbital complications.** *Arch. f. Laryng.*, xxi., No. 3.

204. HEMPEL, H. **Iodides in accessory sinus suppuration.** *Berl. klin. Wochenschr.*, 1908, No. 39, p. 1768.

204A. AXENFELD, T. **Treatment of accessory orbital sinuses by ophthalmologists.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 506.

The first case reported by REBER (164, **Orbital abscess secondary to nasal disease**) was a boy who had had orbital pain, swelling, and proptosis of the right eye for three months. After incision and drainage, in spite of the long duration, there was return to the normal. A large bean had caused a purulent rhinitis, with involvement of the ethmoid cells and rupture into the orbit. The second case was a young woman who developed purulent frontal sinusitis two weeks after an attack of grippe. The lids of the same side were swollen, cornea steamy, nerve head congested and blurred. Orbital exploratory incision revealed pus in which the streptococcus was found. Recovery was uneventful. ALLING.

RISLEY (165, **Binocular exophthalmos. Recovery**) observed a colored woman of forty who showed a proptosis with severe frontal pain first on the right side, then one month later on the left. There were no symptoms of Graves' disease, syphilis, or neoplasm. A skiagraph showed no abnormalities of the cranial bones or cavities. There was possible but doubtful involvement of the anterior ethmoidal cells. The fundus was normal except for paleness of the disks, and vision was $\frac{6}{20}$. Under mercury and iodid of potassium the exophthalmus with the other symptoms disappeared. Risley thinks the most reasonable diagnosis to be a gumma of the base of the brain interfering with the return flow in the ophthalmic vein. ALLING.

EVANS (166, **Topographical anatomy of the sinuses and orbit**). Orbital lesions may originate from contiguous anatomical structures by (*a*) direct infiltration or permeation of tissues, (*b*) extension along lymphatics; (*c*) thrombophlebitis. The author gives a summary of the anatomy of the sinuses with reference to the orbit and its contents, and follows with diagrams of the venous communication of the orbit with the intracranial sinuses. An adequate bibliography is appended. T. HARRISON BUTLER.

EVANS (167, **Ocular and orbital complications of accessory sinus disease**). The paper which is chiefly clinical gives an account of cases of ocular trouble following suppurative

sinusitis, grouping his remarks under the very sinuses involved.

MAXILLARY ANTRUM disease may give rise to lachrymal abscess and to orbital cellulitis.

FRONTAL SINUS disease causes displacement of the globe, headache, and asthenopia.

ETHMOIDAL disease. The *anterior* cells cause orbital abscess on the nasal side and lachrymal suppuration. The *posterior* cells give rise to more complicated symptoms. Most cases complain of loss of sight in one eye and pain around and behind the eye. There is often a central scotoma, or a defect of the fields on the temporal or nasal side. The fundus findings may be nil and the case may be indistinguishable from one of ordinary retrobulbar neuritis, except for the nasal condition. Cases which present irregular defects of the field are more typical of sinus disease, and are generally due to compression of the optic nerve. Other cases (scotomatous) are more probably of toxic origin. When due to compression the recovery of vision after treatment of the sinus is very prompt.

SPHENOIDAL SINUSITIS causes identical symptoms with those just detailed.

Evans concludes by advising that in all cases of retrobulbar neuritis the possibility of a nasal origin should be borne in mind. A mere inspection of the nose is insufficient, the cases should be examined by a competent rhinologist, for preliminary resections of the turbinals, etc., may be necessary before the focus of suppuration can be detected. Many patients do not recover after treatment of the sinusitis.

T. HARRISON BUTLER.

ST. CLAIR THOMSON (168, **Frequency of orbital manifestations of nasal sinusitis**). Diseases of the nasal accessory sinuses are frequent causes of orbital cellulitis. The author points out that the nose is often not examined at all, and one still reads of orbital abscesses coming on "spontaneously." Many cases of thrombosis of the cavernous sinus also originate from a nasal suppuration, in fact Thomson thinks the majority have this origin. He describes cases of orbital inflammation and sinus thrombosis illustrated by photographs. Thomson has a paper on the same subject giving more minute

anatomical details in the *Ophthalmic Review* for October, 1908. T. HARRISON BUTLER.

KATZ (169, **Treatment of tenonitis**) deplores our helplessness in this affection when of severe, protracted type. Chemosis, pain, and diplopia disappear rapidly under compresses of bichloride 1:5000. Suppurative tenonitis may follow perforation of a maxillary empyema into the orbit, or complicate partial, traumatic, purulent choroiditis.

RUS (170, **Orbital phlegmon due to dacryocystitis. Cure**) observed this rare condition with burrowing of pus backwards from the lachrymal sac. Dense opacities of the vitreous cleared up and revealed one or two retinal hemorrhages.

HOFFMANN (171, **Relation of orbital phlegmon to sinusitis**) notes that the latter may give no symptoms and orbital complications are often the first sign of an involvement of the accessory sinuses. Frontal sinus disease is the most frequent cause of orbital complications; the sphenoid, the least common. Radiography is of diagnostic importance. Exploration of the accessory sinus should be advised wherever we cannot be sure of the nasal origin of the orbital complication.

BIRCH-HIRSCHFELD (172, **Relation of orbital inflammations to accessory sinus disease**) analyzes 684 cases, including 40 of his own. Over 60% of orbital disease is due to sinusitis, generally purulent, from rhinitis, influenza, and so on. Periostitis, abscess, or phlegmon occurs and one may lead to the other. Optic nerve involvement must be looked for by examination of color fields and central color perception, especially. Antrum empyema is a frequent source of blindness by orbital thrombophlebitis. Sphenoid empyema frequently leads to blindness and death. Primary orbital phlegmon is unfavorable, as thrombophlebitis is frequent, and death takes place in 17%. Signs of orbital suppuration indicate an incision along the inner margin detaching the periosteum and exposing the frontal and ethmoid cells; Kroenlein's operation may be of service.

KUHNT (173, **Relation of nasal and ocular disease**) notes the frequent involvement of eye and orbit dependent on direct infection via lachrymal passages, mechanical compression by dilatation of neighboring cavities, sensory reflexes, and disturbances of blood and lymph circulation. The frontal sinus

affects the lachrymal passages by preference. Phlegmon of the sac generally originates in the nose. Ethmoid and antrum may be infected from a pus sac. Resection of the middle turbinate is often advantageous. Epiphora, lid œdema, blepharospasm, unilateral blepharitis, and phlyctenulae may be due to nervous disturbances. Trachoma and other lid troubles improve if the nose is treated at the same time. Yellow oxide ointment works well. Uveal inflammation depends largely on intranasal conditions, as does premature senile cataract. Sinus empyema causes contraction of visual field in at least $\frac{1}{6}$ of the cases. Adenoids and carious teeth are responsible for many eye lesions. Kuhnt does not approve of Axenfeld's suggestion to perform Kroenlein's operation for exploration in deep-seated indeterminate orbital inflammation.

Onodi (174, **Relation of nasal and ocular disease**) notes that the optic nerve may be in relation with a posterior ethmoid cell instead of with the sphenoid, or may run free in the sphenoid sinus. It is then exposed to injury during intranasal operation. Oculomotor and trigeminal lesions are often due to lateral extension of the posterior cells into the small and large wing of the sphenoid where they are in relation with the trochlearis and abducens and cavernous sinus.

The nasal finding may be negative, as in case of latent empyema, or obstruction of ostia by polyps. Acute suppuration in posterior cells and acute optic neuritis may disappear under local treatment, but surgical procedures are indicated in case of orbital and intracranial complications. The sphenoid rarely becomes diseased alone; the ethmoid cells are generally found diseased also.

Brueckner (175, **Relation of eye and nose in disease**) studied mainly cases of lachrymal disease and chronic conjunctivitis which did not clear up under treatment. Atrophic rhinitis, hypertrophy, and polyps were the most frequent anomalies; deviations and spurs, less so. Adenoids are not important. In atrophic processes there is an enlargement of the valve of Hasner, allowing infection from the nose. Cocain may relieve pain and diplopia due to frontal sinus disease. In ten cases of unilateral optic neuritis, examination of the nose failed to reveal any disease of the posterior cells.

Chiari and Marschik (176, **Disease of orbit and accessory sinuses**) report 5 cases treated intranasally—3 cured, 2 improved. Mild orbital symptoms such as lid œdema, chemosis, and emphysema disappeared. Blindness of four weeks' standing, due to optic atrophy, was not improved. In 10 other cases the external operation was performed either on account of unsatisfactory result of intranasal treatment, severe orbital symptoms, or involvement of the globe. Definite retinal or nerve lesions always indicated the orbital operation, as do diplopia and exophthalmus. In this series there were 15 cures and 1 death. Angiosarcoma of orbit simulating frontal sinus ectasia was observed.

Newolina (177, **Shot wound of orbit**) noted external ophthalmoplegia lasting for weeks and gradually recovering. The shot had probably injured the trunk of the oculomotor and trochlear. The optic nerve was also injured. The shot lay between the globe and the inner wall of the orbit without affecting cavernous sinus or Gasserian ganglion.

Williams (178, **Bullet wounds of orbit**) reports three cases in which both eyes were blinded by a shot through the temples; one, in which an eye was destroyed by an arrow, and four non-penetrating wounds with more or less complete loss of sight. In two cases, traumatic retinitis was the cause of temporary blindness.

Pechin and Deschamps (179, **Orbital injury and alternate hemiplegia**) noted left ptosis and unconsciousness following a thrust with an umbrella into the left orbit. Right facial paralysis and right hemiplegia developed with return of consciousness. Penetration of the cranial cavity had injured the artery of the fossa Sylvii or the cerebral peduncles.

Dubois de Lavigerie (180, **Spontaneous luxation of globe**) noted this accident in a man of sixty-five. It had happened several times a year for five years. The eye can be replaced by the patient but remains tender for some time. Both globes are prominent but there is no other lesion.

Natanson (181, **Microphthalmus and anophthalmus congenitus with orbital cysts**) connects the cysts with rudimentary development of the eye. They may fill the entire orbit or be so small as to escape detection, are generally unilateral, but both eyes may show developmental anomalies. In

microphthalmus the cyst connects, not with the vitreous cavity but with the space between retina and pigment layer of the eye, the remnant of the primitive optic vesicle. In clinical anophthalmus a rudiment of the globe is found connected with the orbital prolongation of the cyst.

Basso (182, **Tumors of the orbit. Kroenlein operation**) removed from the apex of the orbit a fibrosarcoma the size of a bean which had shown a decided shadow in the radiograph.

Lewit (183, **Operative removal of orbital exostosis**) found a hard mass the size of a pigeon's egg on the upper inner wall of the right orbit of a healthy man, aged twenty-eight years. It had been noticed for about a year, and was removed without much difficulty although rather firmly adherent, especially to the upper wall and in connection with the frontal sinus by a pedicle which had to be cut through.

Fehr (185, **Lymphangioma cavernosum orbitæ**) removed a tumor by Kroenlein's operation, and blunt dissection of the mass, the size of a plum, situated within the muscle funnel in a capsule about the nerve. Abducens paresis due to traction cleared up after a while.

Koster (186, **Spontaneous cure of sarcoma (?) of orbit).** In a youth of sixteen a nodule could be felt under the upper lid, and in four weeks exophthalmus and lid œdema without inflammation developed. An excised mass was diagnosed as sarcoma, but on opening up the orbit a diffuse infiltration of the zygoma and frontal bone was found so that radical removal was impossible. Seven weeks later the tumor was found to be much smaller and the globe was in a normal position. Iodides which had been given before operation in small doses could hardly have brought about this cure.

Wagenmann (187, **Recurrence of melanosarcoma eleven years after enucleation**) noted pain, vertigo, and headache in a man of sixty-seven and removed a black tumor the size of a cherry. After nine months it was necessary to eviscerate the orbit radically. No relapse.

Watanabe (188, **Intravascular endothelioma of orbit**) reports the smooth removal of an angioma of the upper lid and orbit in a child of six months. Orbital relapse within a year. Diffuse masses removed as completely as possible. The tissue resembled that of the lachrymal gland with round

cells and many vessels. No relapse after one and one-half years.

LODATO (189, **Cavernous angioma of orbit**) describes concentric elastic fibres in the capsule of an angioma. Many mast cells were found in the interalveolar septa, particularly in the peripheral portions of the growth. The septa were composed mainly of elastic fibres.

EWETZKY (191, **Significance of orbital teratoma**) describes an enormous tumor filling the orbit and spreading its walls so as to cause marked asymmetry of the face, of elastic consistency and fluctuating. The eye was normal and protruded excessively, finally becoming atrophic from exposure and pressure. Repeated operations finally removed the growth, and the orbit filled up with a bony mass. The histology of the extirpated masses indicated orbital encephaloma.

SCHWALBACH (192, **Inflammatory exophthalmus**) noted protrusion of one eye in a man of fifty. Three months later the other eye began to protrude without ascertainable cause. There was marked local and general reaction, fever, and cachexia. Injections of Deutschmann's serum resulted in a cure after several relapses.

BERTRAM (193, **Congenital bilateral exophthalmus**) saw a child, eight days old, with marked exophthalmus dating from birth. In paroxysms of crying, the right eye alone was luxated in front of the lids. There was an indication of peaked skull. There was premature synostosis diminishing the space of the orbit, and excessive size of brain and distension of venous sinuses explained the luxation. No lues or tuberculosis. The child died some weeks later.

BEAUVOIS (194, **Exophthalmus in maxillary sinusitis of the new-born**) noted the sudden development of left-sided exophthalmus in a child aged fifteen days, which disappeared after purulent nasal discharge lasting six days. The pus contained pneumococci, and suppuration of the antrum was the probable cause.

NICOLAI (195, **Case of traumatic enophthalmus**). The enophthalmus developed about a year after cranial injury and came to a standstill. Hemiatrophy of the face, irregularity of the pupil, and a scotoma for green indicated nervous disturbances besides those to be explained by recession of the globe.

TEICH (196, **Traumatic enophthalmus**) reports a case developing two weeks after injury with extensive fracture and temporal dislocation of the zygoma, as shown by radiography. Tearing of tarso-orbital fasciæ and enlargement of the orbit were the probable causes of recession of the globe and of disturbances of motility in the superior and external rectus.

VAN DUYSE (197, **Traumatic enophthalmus**) reports a case following intraorbital hemorrhage due to fracture of the superior maxilla, zygoma, and orbital margin. There were paralysis of the internal and inferior rectus, mydriasis, and paresis of accommodation. Enophthalmus with sinking of the globe came on about three months after the injury. Without excluding simple atrophy of the orbital cellular tissue or tropho-neurotic disturbances, v. Duyse believes there was actual traction by the consolidating blood clot. This would explain the paralysis of the second branch of the trigeminus by compression of the infraorbital nerve in its canal by clot from the torn infraorbital artery. The persistent paralysis of the inferior rectus was probably due to a cicatricial band.

TOBIAS (198, **Treatment of exophthalmic goitre**) distinguishes between primary, idiopathic, genuine Graves' disease, and the secondary or spurious form. In the glandular variety of the former there is marked hypertrophy of the thyroid, while in another the nervous symptoms are the principal element. Pseudo-Basedow may develop on a basis of thyroid disease or in the course of systemic affections.

OHLEMANN (199, **Exophthalmic goitre**) noted his own symptoms, which were mainly due to circulatory disturbances. Pressure on the jugular from the enlarged thyroid caused œdema of the lids, epiphora, pseudo-pterygium, and finally exophthalmus. The main indication is the reduction of the thyroid. In fresh cases, cold works well. Extirpation is generally the most effectual remedy. Iodides act well.

PREYSING (200, **Perforation of orbit in sinusitis due to scarlet-fever**) notes that this complication is characteristic in scarlet fever. Septic fever, swelling of cervical glands, otitis media with mastoid involvement, and finally lid œdema with a soft tumor at the inner angle are the typical symptoms. Surgical treatment is indicated.

RICK (201, **Osteoma of frontal sinus. Kuester's operation**) performed Kuester's osteoplastic operation for a protrusion at the inner upper angle of the orbit in a man of eighteen. Exophthalmus down and forwards. No limitation of motility. The growth proved to be an ivory exostosis; the mucosa of the frontal sinus was degenerated and polypoid. Tumors, mucocele, suppuration had been excluded by radiographic and other examinations.

STERN (202, **Anomaly of ethmoid labyrinth**) noted marked broadening of the root of the nose of a girl of twelve. Pupillary distance, 66*mm*. Relative convergence showing spreading of ethmoid cells far back.

HEMPEL (204, **Iodides in accessory sinus suppuration**) notes good results from this drug before deciding on surgical intervention.

AXENFELD (204A, **Treatment of accessory orbital sinuses by ophthalmologists**) thinks that the limits of ophthalmic surgery should be extended to include the accessory sinuses of the face, and lays stress on the importance of operative treatment of these cavities for the ophthalmic surgeon as in many orbital complications operation is satisfactory only if parts of the orbital wall and of neighboring sinuses are removed

XI.—CONJUNCTIVA.

205. ROSENHAUCH. **Development of bacterial flora in the conjunctival sac of the new-born.** *Bull. de l'Acad. de Med. des Sci. de Cracowie*, v., 1908.

205A. ELSCHNIG. **Etiology and treatment of chronic conjunctivitis.** *Deutsche med. Wochenschr.*, 1908, No. 26.

206. REH. **Treatment of phlyctenule.** *Fortschr. d. Med.*, 1908, No. 14, p. 417.

207. LAGRANGE, F. **Spring catarrh.** *Arch. d'opht.*, xxviii., p. 337.

208. PASCHOFF, G. **Spring catarrh.** *Ibid.*, p. 110.

209. POSEY, W. **Vernal conjunctivitis with unusual corneal complications.** *Ann. of Ophth.*, Oct., 1907.

210. NATANSON, Jr., A. **Subconjunctival abscess.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 528.

211. SCHICK, B. **Koplik's spots of the lachrymal caruncle in initial stage of measles.** *Muench. med. Wochenschr.*, 1908, No. 16, p. 883.

212. BONDI, M. **Case of Parinaud's conjunctivitis.** *Wiener med. Wochenschr.*, 1908, No. 18.

213. DEHENNE and BAILLIART. **Five cases of Parinaud's conjunctivitis.** *Riv. Ital. di Ottalm.*, iv., February 2, 1908.

214. BASSERES and POUJOL. **Infectious conjunctivitis of Parinaud.** *Ann. d'ocul.*, xxxix., p. 161.

215. BARTZ, J. **Diplobacillary conjunctivitis.** *Inaug.-Diss.*, Rostock, 1907.

216. WEIGELIN, S. **Microscopic study of streptococcus conjunctivitis in a new-born child.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 163.

217. DUTOIT, A. **Streptococcic ophthalmia in scarlet-fever.** *Zeitschr. f. Augenheilkunde*, xix., p. 341.

218. HOLZBACH, E. **Intrauterine acquisition of ophthalmia neonatorum.** *Monatsch. f. Geb. u. Gynaek.*, January, 1908.

219. ELSCHNIG, A. **Ophthalmia neonatorum.** *Prager med. Wochenschr.*, 1908, No. 1.

220. ADAM, C. **Blenolenicet salve in ophthalmia of adults.** *Muench. med. Wochenschr.*, 1907, No. 43.

221. GREEFF, R. **Prophylaxis and treatment of ophthalmia neonatorum.** *Therap. d. Gegenw.*, No. 1, 1908, p. 27.

222. MOISSONNIER. **Atypical case of gonococcic conjunctivitis.** *Ann. d'ocul.*, cxxxix., p. 123.

223. PAPARCONE, E. **Conjunctival reaction of diphtheria and xerosis bacilli.** *Arch. di Ottalm.*, xv., November 8, 1908.

224. GREEFF, FROSCH, and CLAUSEN. **Studies on development of trachoma.** *Arch. f. Augenheilkunde*, lix., p. 200.

225. GREEFF. **Experiments on transmission of trachoma.** *Fortschr. d. Med.*, 1908, p. 114.

226. COHN. **Combating of granular conjunctivitis.** *Vierteljahresschr. f. ger. Med.*, xxxvi., 1908, No. 3.

227. ADDARIO, C. **New surgical cure of trachoma and pannus.** *Arch. di Ottalm.*, xv., November 10, 1908.

228. SELENKOWSKY, J. **Treatment of trachoma with radium.** *Arch. f. Augenheilkunde*, lix., p. 63.

229. FORTUNATI, A., and ESDRA, C. **Histologic changes after radium treatment of trachoma.** *Ann. di Ottalm.*, xxxvii., March, 1908.

230. LUNDSGAARD, K. **Finsen light treatment of trachoma.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 602.

231. CHEVALLEREAU. **Simulated tuberculous pseudo-conjunctivitis.** *Soc. d'opht. de Paris*, January 14, 1908.

232. HANSELL, H. **Acute double miliary tuberculosis of conjunctiva.** *N. Y. Med. Journ.*, March 14, 1908.

233. COHN, S. **Local tuberculin hypersensitiveness of conjunctiva.** *Berl. klin. Wochenschr.*, 1908, No. 17.

234. SONS, G. **Caustic burns. Flap with pedicle for symblepharon.** *Arch. d'opht.*, xxvii., p. 161.

235. GALEZOWSKI. **Lymphectasia of conjunctiva.** *Soc. d'opht. de Paris*, February 4, 1908.

236. DEJONC. **Histology of cavernous lymphangioma of conjunctiva and orbit.** *Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 37.

237. RSCHANYZIN. **Lymphosarcoma of conjunctiva in a child.** *Ibid.*, p. 101.

238. ANCONA, L. **Symmetrical angioma of tarsal conjunctiva.** *Clin. oculist.*, January, 1908.

239. LAUBER, H. **Diffuse melanosarcoma of conjunctiva.** *Zeitschr. f. Augenheilkunde*, xix., No. 2, p. 132.

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241. GUTMANN, A. **Tumors of lachrymal caruncle.** *Zeitschr. f. Augenheilkunde*, xix., No. 16.

242. AURAND, L. **Epithelioma of caruncle.** *Rev. gén d'opht.*, xxvii., p. 145.

243. AUBINEAU. **Primary tumor of palpebral conjunctiva.** *Ann. d'ocul.*, cxxxix., p. 89.

244. GALEZOWSKI, J. **Fibromyxoma of conjunctiva.** *Soc. d'opht. de Paris*, February 4, 1908.

245. POLACK. **Dermo-epithelioma of bulbar conjunctiva.** *Ibid.*, April 7, 1908.

246. PRINCE, A. E. **Treatment of chronic trachoma.** *Journ. Am. Ass.*, April 25, 1908.

247. MACKAY, M. **Prolonged conjunctivitis following Calmette's ocular reaction.** *Boston Med. and Surg. Journ.*, March 12, 1908.

248. LEWIS, F. N. **Ophthalmia neonatorum followed by pyæmia and death.** *Ophth. Record*, January, 1908.

ROSENHAUCH (205, **Development of bacterial flora in the conjunctival sac of the new-born**) found the conjunctival sac sterile at birth with a rapid development of bacteria within a few hours. Staphylococcus pyogenes albus and xerosis bacilli were found regularly. Gonococcic infection generally takes place some days after birth.

ELSCHNIG (205A, **Etiology and treatment of chronic conjunctivitis**) describes two rare forms: C. Meibomiana, due to hypersecretion in adults over forty and in youthful subjects with acne. Digital expression of Meibomian glands is advised. The second form is due to incomplete closure of the lids and affects the exposed area of conjunctiva, and suggests tyloma. Salves and bandaging at night effect a cure.

REH (206, **Treatment of phlyctenule**) notes that the consecutive scars may greatly affect vision. These inflammatory growths are always due to scrofula. General treatment with inunctions and iron, local analgesics, calomel, iodoform, and atropin are advised.

LAGRANGE (207, **Spring catarrh**) reports a case in a boy of five years. There was marked keratitis, tarsal granulations. and large numbers of eosinophile cells in the secretion and granulations.

PASCHOFF (208, **Spring catarrh**) believes that Ehrlich's mast cells are migrating lymphocytes with granular basophile protoplasm. Bacilli of the pseudo-diphtheria group are often present.

POSEY'S case (209, **Vernal conjunctivitis with unusual corneal complications**) was marked by progressive irregular infiltration of the cornea resembling hyaline degeneration. Vessels developed from the limbus. Caustics used in treatment may have caused the latter development.

NATANSON (210, **Subconjunctival abscess**) could find no etiological cause for this condition. Staphylococcus aureus was found.

SCHICK (211, **Koplik's spots of the lachrymal caruncle in initial stage of measles**) noted in the initial stage of measles a swelling of the caruncle with numerous grayish nodules. The rest of the palpebral conjunctiva was red and swollen but there were no spots.

BONDI'S (212, **Case of Parinaud's conjunctivitis**) case developed a corneal infiltrate on the sixteenth day. This was incrusted with lead from eye washes. Many of the cases reported were those of children.

DEHENNE and BAILLIART (213, **Five cases of Parinaud's conjunctivitis**) find the etiology obscure. Five undoubted cases are reported which suggest a constitutional disease with primary local involvement.

BASSERES and POUJOL (214, **Infectious conjunctivitis of Parinaud**) report two cases, in one of which there was a marginal ulcer with perforation and consecutive anterior synechia. Optical iridectomy was made. Of 50 previously reported cases, only three had corneal involvement.

BARTZ (215, **Diplobacillary conjunctivitis**) finds this the most common form in Rostock. Corneal ulcer is common, often with hypopyon. In many cases there was a history of injury. Zinc is effective.

WEIGELIN (216, **Microscopic study of streptococcus conjunctivitis in a new-born child**) notes that this form may

closely resemble gonorrhœal conjunctivitis. There was no streptococcæmia. The infection was due to puerperal streptococcic sepsis of the mother.

Dutoit (217, **Streptococcic ophthalmia in scarlet-fever**) observed this condition on the basis of a burn. It led to severe purulent conjunctivitis and keratitis and finally to staphyloma. The streptococci probably came from the nose.

Holzbach (218, **Intrauterine acquisition of ophthalmia neonatorum**) notes that children were often born with florid blennorrhœa. In his case the bag of waters had ruptured three days before delivery. The vaginal secretion contained gonococci and both eyes of the child showed large corneal ulcers.

Elschnig (219, **Ophthalmia neonatorum**) found gonococci in 21, streptococci in 5, staphylococci in 6, diplococcus pneumoniæ in 1, xerosis bacilli in 3, of 41 cases of ophthalmia neonatorum. Five cases were sterile. Gonococci never disappear from the conjunctiva as long as purulent secretion persists. Nitrate of silver is the only reliable antiseptic. Irrigation with weak permanganate solutions is valuable.

Adam (220, **Blenolenicet salve in ophthalmia of adults**) uses this ointment, which sets free acetate of alumina, and obtained rapid cessation of secretion. Corneal erosions are quickly cured. 10% salve is used at first and then reduced in strength. Adam reports more good results with this treatment especially in corneal ulcer. Vessels form very sparsely and the resulting scars are very thin. (Kipp warns against the use of blenolenicet salve and has seen very serious complications from its application. P. H. F.)

Greeff (221, **Prophylaxis and treatment of ophthalmia neonatorum**) claims that this is curable in every case. ¼% solutions, in instillations, are quite strong enough for prevention. In treatment, 1% solutions are to be applied by irrigation.

Moissonnier (222, **Atypical case of gonococcic conjunctivitis**) noted in a patient of fourteen, œdema of upper lid with a small ulcer near the sclero-corneal margin. The conjunctiva of the upper lid was red, swollen, and velvety, with several whitish (pseudo-membranous?) dots. Tincture of iodin

led to a cure, after silver and permanganate had been used for some time without effect.

PAPARCONE (223, **Conjunctival reaction of diphtheria and xerosis bacilli**) considers these two forms identical. Neither develops into streptothrix. Virulence is the only differential characteristic. Klebs-Loeffler may cause simple catarrhal, purulent, as well as membranous conjunctivitis. Corneal complications generally lead to loss of the eye. The tears have no effect on this bacillus. Xerosis bacillus is not the specific organism in epithelial xerosis or catarrh.

GREEFF, FROSCH, and CLAUSEN (224, **Studies on development of trachoma**) report inoculations with trachoma granules which developed in ten days, and resisted treatment for months. Cell inclusions could not be produced in anthropoid apes.

GREEFF (225, **Experiments on transmission of trachoma**) was able to inoculate a sound eye with trachoma which developed typically in three days. The secretion contained Greeff's micro-organism.

COHN (226, **Combating of granular conjunctivitis**) notes the great infectiousness of trachoma in families and schools and urges rigid prophylaxis.

SELENKOWSKY (228, **Treatment of trachoma with radium**) reports several cures.

FORTUNATI and ESDRA (229, **Histologic changes after radium treatment of trachoma**) noted very rapid action of copper sulphate after radium had been applied to the conjunctiva without apparent result. The latter alone has no effect but prepares the tissues for rapid action of topics.

LUNDSGAARD (230, **Finsen light treatment of trachoma**) reports several good results in removing follicles. The sessions lasted from ten to fifteen minutes.

CHEVALLEREAU (231, **Simulated tuberculous pseudo-conjunctivitis**) noted marked chemosis of the conjunctiva in a girl of twelve. The limbus was completely covered by the swelling. Tuberculin ophthalmo-reaction was positive, but it was found later that the patient herself had artificially and mechanically irritated the eye. When this was prevented the conjunctivitis disappeared at once.

HANSELL (232, **Acute double miliary tuberculosis of conjunctiva**) describes a case in which the conjunctiva of both

upper and lower lids was the seat of small yellowish nodules resembling trachoma. Tubercle bacilli were present in the scrapings. No constitutional signs of tuberculosis and no enlarged glands. ALLING.

COHN (233, **Local tuberculin hypersensitiveness of conjunctiva**) compares the reaction following repeated ophthalmo-tuberculin instillation with the anaphylaxis in serum disease. Hypersensitiveness of normal subjects is characterized by its appearance not only after local instillations but after the von Pirquet injections as well. The reaction begins on the fifth day and is most marked in three weeks.

DEJONC (236, **Histology of cavernous lymphangioma of conjunctiva and orbit**) examined tissue diagnosed as lymphangioma of the orbit. The clinical picture was that of exophthalmus. The subconjunctival swelling appeared bloody after a blow on the brow. Excision of a small piece of the semilunar fold for examination was followed by marked hemorrhagic exophthalmus and suggillation of the ocular conjunctiva due to infiltration of the cavernous spaces with blood from the wound. Lymphangioma always produces a gradually increasing exophthalmus. On exploration of the orbit it is generally impossible to find any distinct tumor mass as the latter collapses and grows again.

ANCONA (238, **Symmetrical angioma of tarsal conjunctiva**) noted this condition in a female infant, aged two years. Successful operation. Bilaterality has not been observed before.

LAUBER (239, **Diffuse melanosarcoma of conjunctiva**) describes dark brown pigmentation of the conjunctiva of lower fornix, neighboring portion of ocular conjunctiva, entire lower lid, half of semilunar fold, and caruncle, which had begun at the last named point two years previously. In a second case of a man of forty-four, injury was followed in a year by a change of the entire ocular and palpebral conjunctiva into a soft black mass which bled easily. Exenteration of orbit in both cases. Microscopic examination corroborated the diagnosis of sarcoma The globe was normal, and the sclera showed no penetration of tumor cells. Enucleation is indicated in spite of this, however on account of the danger of metastasis if operation is delayed.

EMANUEL (240, **Relation of epibulbar melanotic tumors to**

neurofibroma) finds a marked analogy between these two forms, and believes that nævi may be melanotic fibrous growths.

GUTMANN (241, **Tumors of lachrymal caruncle**) describes a benign papilloma in a child of two and one half years, which recurred once, a similar growth of the semilunar fold in patients aged thirty-one and seven years, and a soft fibroma of the caruncle in a female of twenty-one.

AURAND (242, **Epithelioma of caruncle**). Only four cases of this sort have been previously reported. This case was in a woman of thirty-two. The tumor contained pigment.

AUBINEAU (243, **Primary tumor of palpebral conjunctiva**) noted a growth developing in the conjunctiva of the right upper lid of a woman of fifty-three. There was a second growth on the cornea at a point opposite the first. Both were extirpated and found to be polymorphous endothelioma, resembling both epithelioma and sarcoma.

GALEZOWSKI (244, **Fibromyxoma of conjunctiva**) found this tumor on the temporal part of the upper fornix fold. Similar growths have been observed on the buccal and nasal mucosa.

For home treatment PRINCE (246, **Treatment of chronic trachoma**) recommends a 10% solution of sulphate of copper in glycerine which diluted one drop in 20 of water to be used several times a day. The reviewer has found this very useful.

The patient described by MACKAY (247, **Prolonged conjunctivitis following Calmette's ocular reaction**) had pulmonary and arthritic tuberculous lesions. One drop of Calmette's original solution produced a severe reaction in the conjunctiva for ten days. It then lessened for a week but again became severe so that ten weeks after the test it was still in evidence. ALLING.

LEWIS'S (248, **Ophthalmia neonatorum followed by pyæmia and death**) patient presented the ordinary symptoms of ophthalmia neonatorum with ulceration of the right and haziness of both corneæ. While the eyes were still actively inflamed an abscess appeared over the external malleolus of the right foot, which when opened showed pus containing gonococci. Three other abscesses also appeared in other parts of the body with the same findings. The temperature

chart was that of a case of pyæmia, and the child died at the end of five weeks. The writer thinks that sepsis was produced by the absorption of pus from the conjunctiva.

ALLING.

XII.—CORNEA, SCLERA, AND ANTERIOR CHAMBER.

249. DAVIS, H. M., and HULL, G. **Bacteriology of neuropathic keratitis.** *Brit. Med. Journ.*, Jan. 11, 1908.

250. TODD, F. **Instrument for cauterizing corneal ulcers.** *Ophth. Record*, April, 1908.

251. STERN, J. **New corneal phenomenon in trigeminal anæsthesia.** *Klin. Monatsbl.*, xlvi., No. 1, p. 465.

252. SALUS, R. **Green discoloration of cornea in multiple sclerosis.** *Med. Klinik*, 1908, No. 14.

253. KAYSER. **Blood soaking (Durchblutung) of the cornea.** *Wuerttemb. augenaertzl. Verein. klin. Monatsbl.*, xlvi., No. 1, p. 90.

254. DUTOIT, A. **Cocain in corneal affections.** *Korr.-Bl. f. Schwzr. Aertze*, 1908, No. 6, p. 179.

255. BIETTI, A. **Internal ulcer of cornea in parenchymatous keratitis.** *Ann. di Ottalm.*, xxxvii., May 3, 1908.

256. BARTELS, K. **Pathology of disciform keratitis.** *Inaug.-Diss.*, Rostock, 1907.

257. TSCHIRKOWSKY, W. **Atypical hypopyon keratitis.** *Klin. Monatsbl.*, xlvi., No. 1, p. 77.

258. SALVANESCHI, E. **Treatment of hypopyon keratitis.** *Clin. ocul.*, March, 1908.

259. SATTERLEE, G. **Serious result of ocular tuberculin test.** *Journ. A. M. A.*, June 27, 1908.

260. FRUGINELE, C. **Keratitis gummosa.** *Riv. Ital. di Ottalm.*, 1908, iv., No. 3.

261. RUST, E. **Copper oxide in the cornea.** ARCH. OF OPHTH., xxxvi., No. 2, p. 174.

262. CUPERUS, N. **Relapsing corneal erosion.** *Ned. Tydschr. v. Geneesk.*, No. 1, p. 10, 1908.

263. RUPPRECHT, J. **Multiple tears of Descemet's membrane, intra partum.** *Klin. Monatsbl.*, xlvi., No. 1, p. 134.

264. BIETTI, A. **Tumor-like pannus degeneration; hitherto undescribed corneal change.** *Ibid.*, p. 337.

265. FROST, K. **Operative removal of trachomatous pannus.** *Westn. Ophth.*, 1908, No. 2.

266. STEINER, L. **Migration of sepia in tattooed leucomata.** *Centralbl. f. prakt. Augenheilkunde*, xxxii., p. 104.

267. PLANGE, O. **Transplantation of cornea with preserved transparency.** *Klin. Monatsbl.*, xlvi., No. 1, p. 277.

268. KREIBISCH, C. **Hyperkeratosic nævus of skin, nævus of cornea, and cataract.** *Deutsche med. Wochenschr.*, 1908, No. 21.

269. PETERS, A. **Blue marking of globe due to congenital thinning of sclera.** *Klin. Monatsbl.*, xlvi., No. 1, p. 130.

270. VOSSIUS. **Icteric discoloration of sclera in vitreous hemorrhage.** *Deutsche med. Wochenschr.*, 1908, No. 38.

271. ROENNE, H. **Ulcerative scleritis.** *Klin. Monatsbl.*, xlvi., No. 1, p. 522.

272. BIETTI, A. **Pathology and complications of scleritis.** *Ann. di Ottalm.*, xxxvii., May 3, 1908.

273. BUNGE. **Oil drop in anterior chamber.** *Klin. Monatsbl.*, xlvi., No. 1, p. 643.

DAVIS and HULL (249, **Bacteriology of neuropathic keratitis**) first give an account of the various theories which have been put forward to explain the keratitis which so often follows lesions of the fifth nerve and of the Gasserian ganglion. Magendie showed that section of the Gasserian ganglion, especially of the anterior part, gave rise to a destructive keratitis, but that when the fifth nerve alone was cut, although the cornea became anæsthetic, no keratitis followed. From these experiments the cause was supposed to be due to the degeneration of *trophic* fibres.

The *trophic traumatic* hypothesis. Saenger supposed that while trophic fibres had much to do with the matter, the exciting cause was central irritation of the nerves. Parsons considers that this trophic and central traumatic hypothesis "explains better than any other the diverse clinical facts which have accumulated." Samuel thinks that lesions of centrifugal and centripetal fibres render the cornea less able to withstand local irritation.

The *vaso-motor* hypothesis. Spalitta thinks that the vaso-constriction which follows removal of the Gasserian ganglion is the cause of the change. If at the same time he removed the cervical sympathetic ganglion so that vaso-constriction could not occur no keratitis supervened. Schiff thinks hyperæmia lasts for twelve days after excision of the ganglion; occasionally, however, it persists and then keratitis can be produced.

The *purely traumatic* hypothesis. Sneller and others believed that if the eye were suitably protected neuro-paralytic keratitis could not occur; they held that neuro-paralytic keratitis was due to foreign bodies not being promptly expelled from the eye owing to corneal anæsthesia.

The *desiccation* hypothesis. This was held by Hippel and others. After trigeminal neurectomy there is a diminution in the secretion of tears and a lessening in the frequency of blinking; as a result the cornea becomes dry and necrosis results. The authors criticise these theories adversely, and then add evidence in favor of the last hypothesis—the *mycotic*. Turner, Krause, and others believe that septic infection has much to do with the degenerative process. The authors have made bacteriological examinations of 21 cases who had undergone the Hartley-Krause operation. Eleven of them developed neuro-paralytic keratitis. In practically all cases the *Staphylococcus aureus* was found; less frequently the streptococcus, pneumococcus, xerosis and proteus. In addition all the keratitis cases contained a special bacillus which the authors designate the *Bacillus X*. It measures 2.0μ x 0.6μ. It is homogeneous and does not form spores. It is non-motile and has no flagella. It stains with all ordinary dyes and is Gram positive. It grows readily on all ordinary media. It first produces acid, then alkali. It is very like the pseudo-diphtheritic bacillus (and therefore very like the xerosis bacillus-*Abstracter*). The authors think that whatever part *Bacillus X* plays in the initial stages of neuro-paralytic ulceration, staphylococci carry on the work of destruction. The *Bacillus X* was found in 30% of normal eyes. Experiments upon monkeys made by Sir V. Horsley seemed to show that to produce neuro-paralytic keratitis three factors must be present:

(1) Removal of the Gasserian ganglion, (2) the presence of *Bacillus X*, (3) the eyelids must be open. Monkeys when neurectomized and inoculated with cultures of *Bacillus X* did not develop keratitis so long as the eyelids were kept sewn up.

An adequate Bibliography is given and a micro-photograph of *Bacillus X*. T. Harrison Butler.

The instrument described by Todd (250, **Instrument for cauterizing corneal ulcers**) consists of a copper bulb mounted on a handle and having a platinum point and in another place a blunt protuberance. When heated to a cherry red it wil remain hot enough to cauterize for several minutes. It has the advantages over the electro-cautery in its lightness and simplicity. Alling.

STERN (251, **New corneal phenomenon in trigeminal anæsthesia**) noted alternate and often sudden appearance and disappearance of punctate opacities of the corneal epithelium, in both eyes, with stippling of the surface. The patient was a feeble young woman with supraorbital hyperæsthesia and completely anæsthetic corneæ. No hysteria. The opacities disappeared when the lids were kept closed, and with the anæsthesia of the cornea were cured by tonics, strengthening food, and so on, in two months.

SALUS (252, **Green discoloration of cornea in multiple sclerosis**) believes that localization of this stain at the limbus indicates that it develops from deep-seated marginal vessels, probably those from Schlemm's canal, by slow filtration by which hæmoglobin is changed into hæmosiderin. The abnormal condition of Schlemm's canal is probably due to trophic disturbances dependent on multiple sclerosis. (The blood staining of the cornea depends on a chemical change requiring the presence of an acid reaction in the tissue. This explains the localization of the stain, its relation to absence or propinquity of vessels, and the manner in which it clears by absorption. P. H. F.)

DUTOIT (254, **Cocain in corneal affections**) condemns use of cocain as an analgesic in inflammatory conditions, as it seriously interferes with tissue regeneration and causes necrosis, while it has little effect on pain in the presence of inflammatory reaction. Its use should be limited even in removal of foreign bodies.

BIETTI (255, **Internal ulcer of cornea in parenchymatous keratitis**) describes the case of a man of sixty. Corneal epithelium was completely preserved. There was a deep central yellowish infiltrate, surrounded by a blue zone, *2mm* wide, and a peripheral ring infiltrate. There were vessels in the depth of the cornea, deposits on Descemet, nodular exudates on the iris, and occlusion of the pupil. V-perception of light. No fundus reflex. Tuberculin test negative, as was biological experiment, in spite of which Bietti made a diagnosis of probable tuberculosis. Enucleation. Examination showed iridocyclitis, parenchymatous keratitis, internal ulcer of cornea. No tuberculosis found.

BARTELS (256, **Pathology of disciform keratitis**) examined

sections in a case ending fatally by pleuro-pneumonia. There was necrotic sequestration without any inflammatory reaction whatsoever. There was an exudation on the posterior surface of the cornea quite separate and probably independent. No sign of ectogenous infection. The affection was probably a form of herpes corneæ.

Tschirkowsky (257, **Atypical hypopyon keratitis**) describes staphylococcus infection with ulcer formation gradually assuming a necrosing sequestrating form of hypopyon keratitis. A circular hole 5*mm* in diameter with steep edges formed in the cornea, but some sight was preserved by conjunctival flap. Staphylococci were found in the form of sarcina, a very virulent type.

Salvaneschi (258, **Treatment of hypopyon keratitis**) reports 34 cases in which atropin, subconjunctival injections of bichloride, and actual cautery applied to the ulcer worked well. Eserin is dangerous on account of the ciliary irritation it causes. Antipneumococcus serum may be used when the diplobacillus of Fraenkel is present, but is less useful in mixed infections. Instillation of the serum is advisable as a prophylactic measure in superficial corneal lesions.

Satterlee (259, **Serious result of ocular tuberculin test**) noted severe reaction with vascular keratitis and iritis, apparently tuberculous, in a girl of eighteen with enlarged glands.

Fruginele (260, **Keratitis gummosa**) reports a case of nodular keratitis coming on twenty-five years after initial specific lesion. Antiluetic treatment resulted in complete cure, only a small central opacity remaining.

Rust (261, **Copper oxide in the cornea**) notes that the shower of sparks seen in short circuits and irregular contacts of trolley wires consist of incandescent flakes of copper oxide. They often get into the cornea as foreign bodies.

Cuperus (262, **Relapsing corneal erosion**) lays stress on the importance of continuing pressure bandage until the epithelium is fully restored and fluorescin fails to give any stain. Apparent smoothness of the surface is not enough, and a superficial ulcer may develop without renewed injury. Curettage may be necessary for definite cure.

Rupprecht (263, **Multiple tears of Descemet's membrane**)

describes narrow striations irregularly scattered on the posterior surface of the cornea in a child, one day old, whose eye had been compressed by forceps during birth. Microscopically, tears and rolling in of Descemet's membrane were found.

BIETTI (264, **Tumor-like pannus degeneration, a hitherto undescribed corneal change**) found this tissue, consisting of connective tissue and round cells in an eye blind of iritis and secondary glaucoma, which was exposed by defective lids. The new-formed tissue lay between Bowman's membrane and the anterior epithelium.

FROST (265, **Operative removal of trachomatous pannus**) advises the detachment of the vascular layer from the free margin toward the limbus where it is removed with scissors. In extreme cases it may be split at the centre with a Graefe knife and peeled off from the sound cornea. No details of cases are given by Frost.

STEINER (266, **Migration of sepia in tattooed leucomata**) thinks the irregular distribution of particles of India ink after tattooing depends on phagocytosis in some cases. Small inflammatory foci develop and phagocytes attracted by chemotaxis carry the granules from other parts of the leucoma.

PLANGE (267, **Transplantation of cornea with preserved transparency**) operated on a dense corneal scar due to lime burn. A sliver of clear cornea from the phthisical fellow eye was removed with a narrow knife and laid on the deeper layers of the affected cornea after the surface had been split by crucial incision and then scraped off. Disinfectants were avoided, but cocain and contact with forceps did not seem to affect the flap, which healed and became fairly transparent on its new base which was well vascularized.

KREIBISCH (268, **Hyperkeratosic nævus of skin, nævus of cornea, and cataract**) thinks both these conditions, associated with cataract and marked fundus changes in both eyes, depend on analogous disturbances of the epithelial rudiment (Anlage) of the skin and eye.

PETERS (269, **Blue marking of globe due to congenital thinning of sclera**) notes the resemblance with the uvea which is seen through a staphyloma. The condition does not depend on vascular changes or pigmentation, but is due to increased

translucency and perhaps thinning. It was noted in four generations.

Vossius (270, **Icteric discoloration of sclera in vitreous hemorrhage**) noted a yellowish staining due to diffusion of blood, quite like that noted in blood-stained corneæ.

Roenne (271, **Ulcerative scleritis**) saw this complication in nodular scleritis, with marked tendency to relapse. The ulcers were dirty, deep, and resembled miniature intestinal tuberculous ulcers, although both tuberculosis and lues could be absolutely excluded in this case, and iodides had no effect. Local treatment with salicylates internally healed the ulcers, each time, in several weeks.

Bietti (272, **Pathology and complications of scleritis**) reports a case of the rare annular form. Uveitis which is generally considered the primary affection in these cases was but slightly marked in comparison with the scleral lesions, and the infiltrations in the iris and ciliary body were decidedly circumscribed. Scleral injection on the temporal side of the globe is the first sign. Characteristic nodules in the depth of the cornea and epitheloid round cells and giant cells in the accumulations in the anterior chamber strongly suggested tuberculosis.

In Bunge's case (273, **Oil drop in anterior chamber**) of penetrating corneal wound, salve had been rubbed in and evidently got into the anterior chamber, where it remained at the highest level in all positions of the head and coated the iris without obstructing the filtration paths and causing glaucoma as might have been feared.

Sections XIII–XVIII. Reviewed by Dr. NICOLAI, Berlin.

XIII.—LENS.

274. Gifford, H. **On Mittendorf's lens spots.** *Ophth. Record*, March, 1908.

275. Scalinci, N. **Physical chemistry of the lens.** *Arch. di Ottal.*, xv., No. 12, 1908.

276. Isakowitz. **Diffraction in opaque lens (cataracta cerulea).** *Zeitschr. f. Augenheilkunde*, xix., No. 5, p. 401.

277. Blumenthal, L. **Operation of total congenital cataract.** *Westn. Ophth.*, 1908, No. 1.

278. Del Monte, A. **Histology of an old congenital capsulo-cortical cataract.** *Arch. di Ottalm.*, xv., No. 7, 1908.

279. CALDERARO. **Rare form of Morgagnian cataract.** *Clin. ocul.*, March, 1908.

280. WITALINSKI. **Cataracta nigra; etiology, micro-chemistry, prognosis.** *Ophth. Sec., Xth Congr., Pol. Aerzte*, etc., Lemberg, 1907.

281. POSSEK. **Theories of causation of senile cataract.** *Wiener klin. Wochenschr.*, 1908, No. 34.

282. SMITH, H. **Treatment of immature cataract.** *Lancet*, August 15, 1908.

283. MACHEK. **Recession of far point in incipient cortical cataract.** *Zeitschr. f. Augenheilkunde*, xix., p. 330.

284. KITAMURA, S. **Cataract operation in diabetics.** *Klin. Monatsbl.*, xlvi., p. 577.

285. ANGIOLELLA, D. **Collapse of sclera during cataract extraction.** *Ann. di Ottalm.*, xxxvii., Nos. 3–5, 1908.

286. HAESSIG. **Last 500 cataract extractions.** *Zeitschr. f. Augenheilkunde*, xix., p. 71.

287. MENGELBERG. **After-cataract operation with Stilling-Weill harpoon.** XXI*st Congr. Rhein-Westph. Augenaerzt.*, Bonn, 1908.

288. STRAUB. **Cataract extraction in the capsule.** *Niederl. Ophth. Ges.*, 1908.

289. KNAPP, A. **Cataract extraction in the capsule. Visit to Maj. Smith.** ARCH. OF OPHTH., xxxvii., No. 1, p. 13.

290. WEILL, G. **Secondary cataract operations.** *Zeitschr. f. Augenheilkunde*, xix., p. 126.

291. NAPP, O. **Bacteriology of conjunctiva after aseptic bandaging of cataract cases.** *Ibid.*, xviii., p. 198.

292. MARKOWSKI. **Subconjunctival injections of potassium iodide in incipient cataract.** *Ophth. Sec. Pol. Congr.* X., Lemberg, 1907.

293. ARMAIGNAC. **Spontaneous resorption of lens in adults after trauma or discission.** XXV*th Meeting Soc. franç. d'opht.*, 1908.

294. HESSE, R. **Cataract extractions in Graz Univ. clinic.** *Zeitschr. f. Augenheilkunde*, xviii., No. 1.

295. KRAUSS, W. **Question of reclination.** *Ibid.*, xix., No. 32.

296. KUHNT, H. **Zonulotomy.** *Ibid.*, p. 22.

297 ULBRICH. **Senile cataract with secondary glaucoma.** *Ibid.*, xviii., No. 2.

298. RUPP, A. **Rupture of globe with subconjunctival luxation and with extrusion of lens.** *Wiener klin. Rundsch.*, 1908, p. 35.

299 TRUC. **Reclination of cataract with preliminary iridectomy.** *Ann. d'ocul.*, cxxxix., May.

300. PUCCIONI, G. **Complications in cataract operations.** *Clin. ocul.*, February, 1908.

301. KRAEMER, R. **Clinical picture of "phakocele."** *Arch. f. Ophth.*, lxviii., No. 1.

302. ISAKOWITZ. **Coloboma lentis after tear of zonule.** *Arch. f. Augenheilkunde*, lix., No. 4, p. 372.

303. NATHANSON, A. **Disciform opacity of anterior surface of**

lens (Vossius) after contusion and perforating wounds of eye. *Klin. Monatsbl.*, xlvi., No. 1, p. 537.

304. KRUSIUS. **Annular opacities of lens surface after contusion of globe.** *Zeitschr. f. Augenheilkunde*, xvii., No. 6.

305. BERGER, E. **Diseases of teeth and lens affections.** *Soc. odontol. de Paris*, November 24, 1908.

GIFFORD (274, **On Mittendorf's lens spots**) confirms Mittendorf's observations concerning the small spots which are seen at times on the posterior surface of the lens and are in all probability a remnant of the attachment of the hyaloid artery. The reviewer can also testify to their occasional presence. They are best seen with a strong lens in the ophthalmoscope.

ALLING.

ISAKOWITZ [276, **Diffraction in opaque lens (cataracta cerulea)**] excludes absorption and fluorescence in the causation of congenitally blue lenses and attributes the greenish-blue color to diffusion as in glaucoma and normal lenses of the aged.

BLUMENTHAL (277, **Operation of total congenital cataract**) found this condition with nystagmus, alternating strabismus, and iridodonesis in a poorly developed infant of eleven months. Iris tissue thin and ill defined. Pupils contracted and sluggish. Discission: R. without effect; L. extraction. Necrosis of wound lips, occlusion of pupil, and iridocyclitis follow. Retrobulbar abscess and ectasia of operative scar then developed, the latter disappearing after breaking of the abscess.

DEL MONTE (278, **Histology of an old congenital capsulocortical cataract**) noted amyloid degeneration and a netlike complex of degenerated lens fibres. The posterior capsule was wrinkled and inverted. Chalky deposits were seen in the capsule.

CALDERARO (279, **Rare form of Morgagnian cataract**). The patient, aged sixty-three, had complicated cataract with probable chronic choroiditis. Cornea clear. Iridodonesis. Pupil wide and reacted to convergence only. A grayish semitransparent nucleus was seen in the anterior chamber adherent to the lower iris angle and moving with the head. The capsule, 11*mm* in diameter, was perfectly clear and could be seen only with strong focal illumination. T. + 1. Operation successful but V. = light perception was not improved.

There was no trace of epithelium in the anterior capsule and this probably explains the transparency and the absence of shrinking.

WITALINSKI (280, **Cataracta nigra; etiology, micro-chemistry, prognosis**) saw three cases in 1400 of senile cataract. The etiology is obscure as to age, inheritance, or disease.

POSSEK (281, **Theories of causation of senile cataract**) discusses the hypotheses of Becker, Peters, Hess, Roemer, Greeff, and others.

SMITH (282, **Treatment of immature cataract**) advises the removal of immature cataract in the capsule when there are reasons for not waiting. Ripening procedures are unreliable or dangerous. Iridectomy is not always necessary.

MACHEK (283, **Recession of far point in incipient cortical cataract**) notes the unusual development of hypermetropia in peripheral subcapsular opacities in early stages. The volume of the lens diminishes and the curvature changes. Degenerative processes are the cause.

KITAMURA (284, **Cataract operation in diabetics**) reckons 5.5% of cataract, *i.e.*, 0.12% of all eye affections, as diabetic. 112 cases are analyzed. The most frequent complications are iridocyclitis, temporary plus tension, choroidal detachment, post-operative hemorrhage, delayed healing, retinitis, and vitreous hemorrhages. Prognosis is not particularly unfavorable and no variation of technique is indicated.

ANGIOLELLA (285, **Collapse of sclera during cataract extraction**) reports the case of an anæmic woman of forty-three years. On removal of a hard cataract the sclera became inverted but returned to normal position under the bandage in three days. Inflammatory reaction supervened and after twenty-five days the eye was found to be atrophic. Arteriosclerosis, anæmia, and general debility, associated with lack of elasticity of the sclera and diminished resistance of vitreous and uvea, are the probable factors in the few cases on record.

HAESSIG (286, **Last five hundred cataract extractions**) bandages the eyes before operation to detect latent inflammation and to accustom the patients to post-operative conditions. The patients are operated on a table and walk to their beds. Graefe section in scleral margin without conjunctival flap. Iridectomy, capsulotomy, bandage, the second eye being

covered with plaster strips for two days. One day later dressings changed and atropin instilled. Cortical remnants were the most frequent complication. Loss of vitreous was unusual. Open treatment does not favor prompt healing but the bandage should be removed early.

MENGELBERG (287, **After-cataract operation with Stilling-Weill harpoon**) uses Stilling's harpoon-needle, which may have to be fastened, if it does not take hold, by means of a punch forceps.

STRAUB (288, **Cataract extraction in the capsule**) advises this procedure in case of thickened capsule, congenital cataract, and the massive form following iridocyclitis. The advantages are undoubtedly the black pupil and rapid healing, but there is danger of loss of vitreous.

KNAPP (289, **Cataract extraction in the capsule. Visit to Maj. Smith**) describes the Indian method as applied to three thousand extractions in the course of twelve weeks every Spring and Fall. Fifty operations a day are often performed. The eyes are washed with soap and water and irrigated with bichloride. Narrow iridectomy usually precedes. The lens is delivered by pressure of a large squint hook on the cornea, without capsulotomy. Dressings changed after four days. The section lies entirely in clear cornea. In case of prolapse of vitreous a spatula is introduced behind the lens. Iris prolapse and incarceration are the most frequent complications. Secondary operation is never needed.

WEILL (290, **Secondary cataract operations**) uses the harpoon needle of Stilling and has no accidents. There may be some difficulty in withdrawing the instrument on account of the barbs catching, obviated by pressure on the opposite side of the puncture.

MARKOWSKI (292, **Subconjunctival injections of potassium iodide in incipient cataract**) notes improved vision in 4 out of 5 cases. The opacities did not appear to become much clearer.

HESSE (294, **Cataract extractions in Graz Univ. clinic**) reports five hundred cases from Dimmer's clinic. Homatropin, and later eserin, is instilled before operation. Iridectomy is performed only in special cases, such as extreme senility, corpulence, cough, immature cataract, previous loss of one

eye. Flap extraction upward. Bed rest for one day, discharge in fourteen days. Secondary operations in about 7% of uncomplicated cases. Iridocyclitis, corneal opacities, high myopia, glaucoma, detachment, and choroiditis were the most frequent complications.

KRAUSS (295, **Question of reclination**) was obliged to eschew extraction in a case complicated by myopia, iridocyclitis, and detachment, fearing loss of vitreous and hemorrhage. The reclination was not complete and was followed by iridocyclitis with total blindness.

KUHNT (296, **Zonulotomy**) advises cutting the zonule in intraocular irritation due to traction by secondary membranes, cyclitic adhesions, on the ciliary body, and in reclination of cataract, which has again come into use for complicated cases. Membranous secondary cataracts, traumatic forms with degeneration and shrinking, rudimentary and congenital types are also suitable for zonulotomy.

RUPP (298, **Rupture of globe with subconjunctival luxation and with extrusion of lens**) reports two cases in which the lens was extruded from the eye. One was due to a thrust by the horn of an ox, and one to blunt injury from a large piece of board. Infection is the main danger to be guarded against in these cases.

TRUC (299, **Reclination of cataract with preliminary iridectomy**) operated successfully on a man of seventy whose other eye had been lost thirty years previously. First an old trichiasis was cured, then a dacryocystitis treated and iridectomy performed. The cataract was couched. After operation the cautery was again applied. V. was improved from F. at 1.5*m* to $\frac{1}{10}$.

PUCCIONI (300, **Complications in cataract operations**) reports a case of severe post-operative hemorrhage originating from the iris alone, and dependent on nephritic vascular disease.

ISAKOWITZ (302, **Coloboma lentis after tear of zonule**) noted this condition in a blind boy of eighteen. Double iridectomy had been performed at the age of one year for adherent leucoma.

NATHANSON [303, **Disciform opacity of anterior surface of lens (Vossius) after contusion and perforating wounds of eye**]

describes a ring-shaped zone corresponding to the pupillary margin which in a month became quite diffusely opaque, the original ring having disappeared. Marked pain necessitated operation. V = 0.8 with correction.

KRUSIUS (304, **Annular opacities of lens surface after contusion of globe**) reports injury of the globe with a piece of iron followed by a circular opacity of the anterior surface of the lens which could be seen distinctly when the pupil was dilated and was present five weeks after the injury.

BERGER (305, **Diseases of teeth and lens affections**) notes that only the relation of rachitic teeth to zonular cataract has been studied. In tetany there is loss of hair, teeth, nails, and cataract formation. In some cases of tetany of pregnancy there may be cataract only, in others, loss of teeth alone. Parathyroid therapy has been used successfully for the spastic conditions present. Senile cataract and loss of teeth in the aged are, however, independent phenomena.

XIV.—IRIS.

306. FAITH, T. **Traumatic irideræmia.** *Annals of Ophth.*, October, 1907.

307. STEPHENSON. **Persistent capsulo-pupillary membrane and hyaloid artery.** *Ophthalmoscope*, May, 1908.

308. BURT, J. **Acute iritis in rheumatoid arthritis.** *Ibid.*, January, 1908.

309. DUNN, P. **Infective cyclitis and penetrating wounds.** *Brit. Med. Journ.*, No. 11, 1907.

310. LAQUEUR, L. **Studies in the pathology of the pupil.** *Arch. f. Augenheilkunde*, lix., No. 4, p. 327

311. VALOIS. **Case of aniridia.** *L'Ophthalm. provinc.*, 1908, No. 1.

312. LUTZ, A. **Cases of heterochromia of irides.** *Zeitschr. f. Augenheilkunde*, xix., pp. 208, 345.

313. PUCCIONI, G. **Pathogenesis of traumatic and congenital iris cysts.** *Clin. oculist.*, May-June, 1908.

314. ZIMMERMANN. **Typical iris coloboma.** *Arch. f. Ophth.*, lxvi., No. 2, p. 270.

315. CARLINI, V. **Serous traumatic iris cysts.** *Ann. di Ottalm.*, xxxvii., No. 6, 1908.

316. COSMETTATOS, G. **Three cases of microphthalmus.** *Ann. d'ocul.*, cxxxix., p. 96.

317. CHEVALLEREAU. **Iris tumor.** *Soc. d'opht. de Paris*, July 7, 1908.

318. REMBE. **Cysticercus cellulosæ of iris.** *Ophth. Record*, January, 1907.

319. FLEISCHER. **Sarcoma of iris.** *Verein. Wuerttemb. Aerzte*, Tuebingen, May 24, 1908.

320. GROENOUW. **Iris tumor of twenty-three years' standing.** *Centralbl. f. prakt. Augenheilkunde*, xxxii., p. 101.

321. ZIEGLER, S. **History of iridotomy.** *Am. Med. Assn., Sect. Ophth.*, Chicago, 1908.

322. TERRIEN, F. and CANTONNET, A. **Blood elements and etiology of iritis.** *Arch. d'opht.*, xxvii., p. 297.

323. RUPPRECHT, J. **Multiple tears of Descemet's membrane intra partum.** *Klin. Monatsbl.*, xlvi., No. 1, p. 134.

324. PERLMANN. **Traumatic serous cyclitis.** 20 *Vers. Rhein. Westph. Augenaerzt.* Duesseldorf,, February 9, 1908.

325. DENIG, R. **Paracentesis of anterior chamber in anterior uveal disease.** *Ophth. Record*, March, 1908.

326. DE SCHWEINITZ, G. and FIFE, C. **Auto-intoxication and corneal and uveal disease.** *Am. Med. Assn., Sect. Ophth.*, Chicago, 1908.

327. D'ALLESSANDRO. **Tuberculosis of iris diagnosed and treated with tuberculin.** *Clin. ocul.*, May-June, 1908.

The first case described by FAITH (306, **Traumatic irideræmia**) was a perforating wound of the cornea, through which immediately after the injury the iris was entirely pulled out by the attending physician. The eye later became quiet and retained one-third vision. Another case was one of contusion with dislocation of the lens into the vitreous and with the entire iris separated and hanging in shreds. A third case was a corneal wound with total irideræmia and later absorption of the lens with one-half vision. There is usually profuse hemorrhage with increase of tension, and atropin is contra-indicated. ALLING.

STEPHENSON (307, **Persistent capsulo-pupillary membrane and hyaloid artery**). The membrane in the pupillary area of the right eye is beautifully shown in the drawing which accompanies the communication. There were also present on the right side: (1) a persistent hyaloid artery which did not contain blood; (2) an atypical mesoblastic development. The retinal vessels were sheathed in opaque white glistening bands, which in some instances extended for a considerable distance into the fundus oculi almost concealing the vessels. The left eye also showed some remnants of a pupillary membrane. T. HARRISON BUTLER.

BURT (308, **Acute iritis in rheumatoid arthritis**) records a case illustrated by excellent skiagraphs of the affected joints

in which recurrent iritis was associated with exacerbations of chronic rheumatoid arthritis. The iritis was of a mild type, but a posterior synechia formed during one attack. The first onset of iritis was noticed within three months of the first symptoms of arthritis. There was no history of rheumatic fever, and gonorrhœa and syphilis were not present.

T. HARRISON BUTLER.

DUNN (309, **Infective cyclitis and penetrating wounds**). The author objects to the terms sympathetic ophthalmia or ophthalmitis. He suggests that it be called "infective cyclitis." There are three stages: (*a*) the infection period, (b) the incubation period, (c) the period of development. There is always a history of injury to the eye which is first infected, but it may be slight, such as a tattooing operation. The shortest time in which infection of the second eye has appeared is ten days, but usually from three to six weeks elapse. The disease has started after months and even years. Dunn draws a comparison with traumatic tetanus. The time of incubation of Nicolaier's bacillus varies much as does the time of onset of sympathetic cyclitis. The ciliary region and the vitreous form a splendid culture medium for a micro-organism, and in consequence wounds here should be dealt with in accordance with general surgical principles; no general surgeon would suture or close a wound which he suspected might be contaminated with tetanus bacilli. The ophthalmic surgeon always fears that the second eye may sympathize, and so he also should never suture a penetrating wound of the globe. Dunn never sutures the sclerotic or conjunctiva, nor does he replace any part of the ciliary body which may have prolapsed. He always asks himself three questions when called to treat such a wound: (1) Is there evidence that a foreign body is retained? (2) Is the injury too severe to preclude the possibility of recovery? (3) What are the prospects of useful vision being retained? Dunn never considers the question of possible infection of the second eye, for with modern antiseptic methods it is an exceedingly rare event. He never enucleates an eye until he has seen what nature will do. If the central area of the cornea be intact and the iris and lens sound, the surgeon must try to save the eye. In the open treatment of ciliary wounds recovery generally takes

place without any ciliary reaction. This is so even when vitreous has escaped and the ciliary body has prolapsed. Gaping of the wound is a favorable feature and the edges must not be approximated. Tension remains minus for several weeks, and long after sound wound healing has taken place. Mackenzie mentions that he never saw sympathetic cyclitis follow extraction. Dunn says that nearly all extractions in those days suppurated and panophthalmitis never causes sympathetic trouble. Nowadays a common result of sympathetic cyclitis is for the eye to recover full vision, in olden times both eyes were almost invariably lost. The reason is that antiseptics have so far attenuated the infecting organism that even if sympathetic cyclitis supervene it is of a benign type. T. HARRISON BUTLER.

LAQUEUR (310, **Studies in the pathology of the pupil**) reports pupillary conditions after section of the optic nerve, in amaurosis due to hemorrhages, embolism of the central retinal artery, choked disk, retrobulbar neuritis, tabes, optic atrophy, and local disease. Details of meiosis, mydriasis, convergence, light reaction, and consensual reaction are given.

VALOIS (311, **Case of aniridia**) removed a cataract in a case with absence of iris and made use of a convex glass with a stenopaic slit.

LUTZ (312, **Cases of heterochromia of irides**) notes insufficient development or later degeneration of pigment as a cause of this condition. Affections of the sympathetic, cyclitis, cataract, glaucoma, Descemetitis, vitreous opacities, are among the complications.

PUCCIONI (313, **Pathogenesis of traumatic and congenital iris cysts**) reports two cases. The traumatic cyst developed after cataract extraction and was due to inclusion of epithelium in the section up to the iris (Stoelting). Traumatic cysts are inclusion or retention cysts, but the congenital form is probably of retinal epithelial origin and indirectly ectodermal.

ZIMMERMANN (314, **Typical iris coloboma**) noted in the case of a man of twenty-seven, a narrowing of the limbus inward and downward, flattening, and scar formation, with almost complete absence of iris, probably due to inflammation during intra-uterine life.

CARLINI (315, **Serous traumatic iris cysts**) reports two cases

with histological examination. The implantation theory (Rothmund-Masse) applies to pearl cysts but does not explain those with epithelial lining, which are due to direct proliferation of the corneal lining, forming a plug in the wound canal reaching to the prolapsed iris. Serous epithelial and pearl cysts are due to implantation of ectoderm.

Cosmettatos (316, **Three cases of microphthalmus**) found this condition associated with coloboma of iris and cataract, with phthisis bulbi, and with hare-lip, respectively. Histological examination of one case showed inflammation of the mesodermal structures due to infection during intra-uterine life.

Groenouw (320, **Iris tumor of twenty-three years' standing**) describes a tumor which had led to blindness in five years. It was probably a melanosarcoma or melanoma which had become sarcomatous, and early iridectomy and excision of the growth might have brought about a cure.

Rupprecht (323, **Multiple tears of Descemet's membrane intra partum**) reports a case in which traumatism resulted in imbibition of the cornea with aqueous, detachment of choroid and retina. Cases which are now considered as cyclitis were formerly called serous iritis or irido-choroiditis. We must distinguish between these cases and those of hyalitis, or cyclitis with vitreous infection.

Perlmann (324, **Traumatic serous cyclitis**) reports a case with vitreous opacities five weeks after a foreign-body injury of the cornea, without any other ascertainable etiological factor besides trauma.

Denig (325, **Paracentesis of anterior chamber in anterior uveal disease**) reports 12 cases of iritis, cyclitis, interstitial keratitis, chorio-retinitis, and diabetic retinitis, treated by repeated puncture with good results, especially in diminishing pain. A small lance knife is used and the wound reopened in a few minutes.

de Schweinitz and Fife (326, **Auto-intoxication and corneal and uveal disease**) believe there is a connection of this sort, and report good results after regulation of diet. The details are important diagnostically and therapeutically.

d'Allessandro (327, **Tuberculosis of iris diagnosed and treated with tuberculin**) reports the case of a girl of thirteen

in which enucleation had been advised for tuberculosis of the iris. Operation was refused. Tuberculin treatment resulted in a complete cure. T. R. is the best preparation and should be used in early injections of small doses.

XV.—CHOROID.

328. Behse, E. **Anatomy of myopic crescent and choroidal changes.** *Arch. f. Ophth.*, lxvii., No. 3, p. 379.

329. Gebb, H. **Punctate choroidal atrophy and pigmentation.** *Arch. f. Augenheilkunde*, lix., No. 4, p. 383.

330. de Lapersonne. **Rare form of choroidal atrophy in old chorio-retinitis.** *Soc. d'opht. de Paris*, March 10, 1908.

331. Guglianetti, L. **Family vascular sclerosis of choroid.** *Arch. di Ottalm.*, xv., No. 12, 1908.

332. Marbais. **Case of choroideræmia.** *Soc. Belge d'opht.*, Brussels, 1908, No 4, p. 26.

333. Rollet and Aurand. **Experimental study of choroidal infections.** *Rev. gén. d'opht.*, No. 1, 1908.

334. Small C. **Microscopic study of metastatic hypernephroma of choroid.** *Ophth. Record*, No. 2, 1908.

335. Lafon, C. **Metastatic carcinoma of choroid.** *Recueil d'opht.*, xxx., p. 193.

336. Kassapian. **Melanotic sarcoma of choroid invading lachrymal sac and canal.** *Thèse de Lyon*, 1907.

337. Wagenmann, A. **Melanosarcoma; relapse eleven and one-half years after enucleation.** *Klin. Monatsbl.*, xlvi., No. 1, p. 140.

338. Terson, A. **Traumatic subchoroidal and expulsive hemorrhages.** *Arch. d'opht.*, xxvii., p. 446.

339. Cohen, C. **Choroidal abscess behind macula after penetrating wound with percussion-cap. "Hole in the macula."** *Klin. Monatsbl.*, xlvi., No. 1, p. 620.

340. Bull, C. S. **The deep intraocular lesions of infantile hereditary syphilis and tardy syphilis, from the standpoint of general pathology, prognosis, and treatment.** *Med. Record*, April 14, 1908.

Behse (328, **Anatomy of myopic crescent and choroidal changes**) reviews the theories, including the view of Heine of an optic-nerve fold causing pressure between retina and choroid. Histological examination of eleven globes showed that the myopic conus is due to traction, as indicated by strictly circumscribed choroidal atrophy with disappearance of pigment epithelium. Distraction crescents and fold of the optic nerve are rare.

Gebb (329, **Punctate choroidal atrophy and pigmentation**) describes numerous small round yellow deposits in the periph-

ery with small pigment dots, with central scotoma for white and colors. V. = F. at 1–1½*m* with eccentric fixation. Hereditary lues was suspected.

GUGLIANETTI (331, **Family vascular sclerosis of choroid**) reports the case of a man of thirty-two who at the age of twenty-five began to notice loss of sight which rapidly progressed. V = F. at 1*m* O.D., at 3*m* O.S. Fundus showed sclerosis of choroidal vessels most marked about the macula, pigment infiltration of the retina, suggesting the bone-corpuscle figures of retinitis pigmentosa. Vessels and disk normal. A sister of the patient presented the same picture and history.

ROLLET and AURAND (333, **Experimental study of choroidal infections**) got only negative results in inoculations of the choroid of rabbits with oïdium albicans, lepra, typhoid, influenza, and coli communis bacilli; general systemic infection, with abscess of liver but no ocular infection, with actinomyces; while all micro-organisms but actinomyces and influenza bacillus injected into the vitreous caused more or less marked infection, extending in some cases to the general system, but remaining localized and of slight severity in the case of typhoid.

SMALL (334, **Microscopic study of metastatic hypernephroma of choroid**) enucleated an eye after penetrating injury suggesting retained foreign body. Above the disk a tumor was found with well marked yellowish, necrotic centre and a cortex of epithelioid cells. No glycogen was found and there was no history of hematuria and no discoverable tumor in the kidney region.

LAFON (335, **Metastatic carcinoma of choroid**) describes the case of a man of forty-two. The primary cancer was situated at the base of the right lower lobe of the lung. Fifty-six cases are on record.

KASSAPIAN (336, **Melanotic sarcoma of choroid invading lachrymal sac and canal**) reports two cases. In one, the invasion proceeded in the mucosa of the lachrymal passages, while in the other the tumor masses broke through the orbital wall in a recurrence and thus entered the sac.

WAGENMANN (337, **Melanosarcoma; relapse eleven and one-half years after enucleation**) advises exenteration of the orbit in tumors of long standing with slow growth in order

to prevent metastases. In his case enucleation had been performed for choroidal sarcoma and the recurrence took place in spite of apparent limitation of the tumor to the interior of the globe.

COHEN (339, **Choroidal abscess behind macula after penetrating wound with percussion-cap. "Hole in the macula"**) noted the development of a hole in the macula after penetrating wound by a percussion-cap. Histological examination showed œdema and local changes.

BULL (340, **The deep intraocular lesions of infantile hereditary syphilis and tardy syphilis, from the standpoint of general pathology, prognosis, and treatment**) believes that diseases of the uveal tract in young infants, especially iritis and choroiditis, appear more frequently than keratitis as evidence of hereditary syphilis. Iritis rarely exists without choroiditis, and these are not infrequently found at birth. He has never seen active retinitis or optic neuritis at birth, but has observed neuro-retinitis in the atrophic stage. Although infants are said to be free from actual symptoms of syphilis when born, he believes it is not uncommon to find internal lesions immediately if they are thoroughly searched for. In closing his paper he propounds the proposition that it should be a penal offence for syphilitics to marry. ALLING.

XVI—VITREOUS.

341. HEPBURN. **Persistent hyaloid artery.** *Ophth. Soc. U. K.*, March 12, 1908.

342. DE VRIES, W. **Persistent hyaloid artery.** *Ned. Tydsch. v. Geneesk.*, No. 1, p. 18, 1908.

343. LANDOLT. **Periodic hemorrhages into the vitreous.** *Soc. d'opht. de Paris*, iii., No. 10, 1908.

344. KYRIELEIS. **Phlebotomy in relapsing intraocular hemorrhages.** *Klin. Monatsbl. f. Augenheilkunde*, xlvi., No. 1, p. 513.

345. KRAUSS, F. **Persistent hyaloid artery and canal of Cloquet.** *Ophth. Record*, April, 1907.

346. KRAUSS, F. **Recurrent hemorrhages into the vitreous in adolescents.** *Ann. of Ophth.*, January, 1908.

KYRIELEIS (344, **Phlebotomy in relapsing intraocular hemorrhages**) advises this procedure in order to reduce blood pressure and thus guard against repeated hemorrhages. It is not indicated in anæmia, cachexia, or hemophilia.

According to KRAUSS (346, **Recurrent hemorrhages into the vitreous in adolescents**) these cases are usually young men between seventeen and twenty-eight, in depressed health and subject to constipation. The etiology is unknown, although they are probably due to auto-intoxication from intestinal absorption, with increased blood pressure and probably also a weakness or local disease of the blood-vessels of the eye. His own case was in a twenty-six-year-old man who had had six hemorrhages in two and one-half years, involving only the right eye. At the final examination, the vision had improved to normal, although there were numerous opacities in the lower part of the vitreous.

ALLING.

XVII.—GLAUCOMA.

347. HOSFORD. **Buphthalmus with mal-development of iris.** *Ophth. Soc. U. K.*, Nov. 13, 1908.

348. ERDMANN. **Experimental glaucoma and examination of glaucomatous animal's eyes.** *Arch. f. Ophth.*, lxvi., No. 3, p. 391.

349. URIBE Y TRONCOSO. **Chemistry of aqueous in glaucoma.** *Soc. d'opht. de Paris*, January 14, 1908.

350. TERSON, A. **Pathogenesis of primary glaucoma.** *Gaz. des hôp.*, 1908, No. 52, p. 615.

351. TERSON, A. **Pathological analysis and synthesis of glaucoma.** *Arch. d'opht.*, xxviii., No. 11.

352. KNAPP, A. **Histological examination of a case of acute primary glaucoma.** ARCH. OF OPHTH., xxxvii., No. 2, p. 157.

353. SCHNABEL. **Theory of pathological optic-nerve excavation.** *Zeitschr. f. Augenheilkunde*, xix., p. 556.

354. SCHMIDT-RIMPLER. **Pressure excavation and optic atrophy.** *Arch. f. Augenheilkunde*, lix., No. 1.

355. PUSEY, B. **Glaucomatous excavation of the lamina without excavation of disk and marked papillitis.** ARCH. OF OPHTH., xxxvii., No. 2, p. 160.

356. WATANABE, B. **Interpretation of occlusion of iris angle and optic-nerve excavation.** *Zeitschr. f. Augenheilkunde*, xix., p. 109.

357. LAWFORD, J. **Examples of hereditary, primary glaucoma.** *Royal London Hosp. Reps.*, 1908, No. 7.

358. WUERDEMANN, H. V. **Diaphanoscopy in glaucoma.** *Ophth. Rec.*, March, 1908.

359. CHENEY, F. **Treatment of simple glaucoma.** *Ophthalmoscope*, iii., No. 3, 1908.

360. POSEY, W. C. **Treatment of simple chronic glaucoma with meiotics.** *Am. Med. Assn., Ophth. Sect.*, Chicago, 1908.

361. LAGRANGE, F. **Simple sclerotomy in simple chronic glaucoma.** *Soc. d'opht. de Paris*, July 7, 1908.

362. LAGRANGE, F. **Sclerecto-iridectomy and filtering scar in the cure of chronic glaucoma.** *Ibid.*, January 14, 1908.

363. BETTREMIEUX. **Simple sclerectomy.** *L'Echo méd.*, xxii., 1908.

364. HERBERT. **Filtering scar in treatment of glaucoma.** *Ophthalmoscope*, May, 1908.

365. VALUDE. **On irido-sclerectomy.** *Ann. d'ocul.*, cxxxix., May, 1908.

366. ROCHON-DUVIGNAUD. **Conditions of efficacy of irido-sclerectomy.** *Arch. d'opht.*, xxviii., p. 138.

367. HEGNER, C. **Malignant glaucoma following iridectomy; relief for ten years from sclerectomy.** *Zeitschr. f. Augenheilkunde*, xix., No. 6, p. 552.

368. MEYER, A. **Histology of thrombotic retinal apoplexy and secondary glaucoma.** Tuebingen, August, 1907.

369. COPPEZ, H. **Spontaneous rupture of globe in glaucoma.** *Soc. d'opht. de France*, 1908.

370. BIALETTI, C. **Two cases of spontaneous rupture.** *Ann. di Ottalm.*, xxxvii., No. 27.

371. BLESSIG, E. **Glaucoma and systemic disease.** *Petersb. med. Wochenschr.*, No. 34, 1907.

372. ISAACS. **Glaucoma due to swollen cataract.** *Klin. Monatsbl.*, xlvi., No. 1, p. 640.

373. MELLER, J. **Cyclodialysis and its effect on tension.** *Arch. f. Ophth.*, lxvii., No. 3, p. 476.

374. KRAUSS, W. **Cyclodialysis.** *Zeitschr. f. Augenheilkunde*, xx., No. 1, p. 50.

375. MARKLEITNER, I. **Traumatic cyclodialysis.** *Klin. Monatsbl.*, xlvi., No. 1, p. 137.

376. JUDIN, K. **Cyclodialysis: clinical and experimental study.** *Westn. Ophth.*, No. 2, 1908.

377. RUMSCHEWITSCH. **Case of hydrophthalmus.** *Ophth. Gesells. in Kiew*, July 15, 1908.

378. STEPHENSON. **Buphthalmus healed by iridectomy.** *Ophth. Soc. U. K.*, Feb. 13, 1908.

379. STOELTING. **The normal operation in hydrophthalmus. Iridectomy or sclerotomy?** *Arch. f. Ophth.*, lxvii., No. 1, p. 171.

380. CHALUPECKY. **Glaucoma and injury.** *Wiener klin. Rndsch.*, 1907, p. 43.

381. KUSCHEL, J. **Senile sclerosis of hydrostatic-regulation apparatus as predisposing cause of glaucoma.** *Zeitschr. f. Augenheilkunde*, xix., p. 193.

382. KUSCHEL, J. **Acute glaucoma as maximum of glaucomatous disposition.** *Ibid.*, Supplement, p. 45.

383. KUSCHEL, J. **Chronic simple glaucoma as next higher degree of glaucoma tendency.** *Ibid.*, p. 426.

384. VEASEY, C. **Glaucoma in the young.** *N. Y. Med. Journ.*, Jan. 4, 1908.

TERSON (350, **Pathogenesis of primary glaucoma**) discusses heredity, social conditions, occupation, sex, and race. Gout and rheumatism are definite, syphilis a possible factor. Arteriosclerosis and many organic diseases are of importance. High tension seems to be the link. Acute glaucoma is compared with acute œdema without inflammation. The mechanism of hypersecretion is analyzed.

SCHNABEL (353, **Theory of pathological optic-nerve excavation**) recognizes three types: 1. v. Graefe's amaurosis with excavation. 2. Successful iridectomies with consecutive excavation. 3. Plus tension leading to excavation. The clinical features of these types are contrasted.

WATANABE (356, **Interpretation of occlusion of iris angle and optic-nerve excavation**) examined various globes with phthisis following injury, buphthalmus, and glaucoma primary and secondary, as to the conditions at the iris angle and on the disk. The relation is not definite.

WUERDEMANN (358, **Diaphanoscopy in glaucoma**) notes that a small circumlental space determined by transillumination is a factor in glaucoma, predisposing, in congestive states of the root of the iris, to tension.

POSEY (360, **Treatment of simple chronic glaucoma with meiotics**) found that in 110 eyes treated in this way, 80% retained vision or improved, 12% were worse, and 8% did badly. Meiotics give favorable results.

LAGRANGE (361, **Simple sclerotomy in simple chronic glaucoma**) lays stress on correct performance of his section which must go through the ligamentum pectinatum iridis and divide the insertion of the muscle of accommodation, so as to establish a communication between the lymph spaces of the choroid and the anterior chamber. This produces either a filtering scar with thinned sclera, a small subconjunctival fistula, or a cystoid scar. The sclerotomy must be narrow.

LAGRANGE (362, **Sclerecto-iridectomy and filtering scar in the cure of chronic glaucoma**) insists on a filtration cicatrix connecting the suprachoroidal space and the subconjunctival tissues with the chambers of the eye, which reduces tension and cures glaucoma as far as surgery can cure it.

BETTREMIEUX (363, **Simple sclerectomy**) believes this operation can take the place of iridectomy in suitable cases and that it is free from danger.

HERBERT (364, **Filtering scar in treatment of glaucoma**) describes a jagged cut to assure a permanent filtration, the main object of iridectomy.

VALUDE (365, **On irido-sclerectomy**) notes occasional bad results from the operation of Lagrange. Recurrence was noted, requiring meiotics.

ROCHON-DUVIGNAUD (366, **Conditions of efficacy of irido-sclerectomy**) objects to Lagrange's operation that only a wide sclerectomy can assure a filtering scar. This he has proven in several cases.

HEGNER (367, **Malignant glaucoma following iridectomy; relief for ten years from sclerectomy**) reports plus tension after influenza, unaffected by iridectomy. Vision was reduced, the anterior chamber abolished. Two posterior sclerectomies improved the condition and vision rose with diminution of tension, restoration of the anterior chamber, and a cystoid scar. Ten years later the good result was still apparent.

MEYER (368, **Histology of thrombotic retinal apoplexy and secondary glaucoma**) describes hemorrhagic glaucoma as understood by v. Graefe. Three cases are reported in which enucleation was performed, but microscopic examination failed to reveal the relation between hemorrhages and plus tension.

COPPEZ (369, **Spontaneous rupture of globe in glaucoma**) noted this accident in a man of sixty-nine, following intense emotional excitement. Microscopic examination showed that intraocular hemorrhages had preceded.

BIALETTI (370, **Two cases of spontaneous rupture**) reports this occurrence in two cases, three, and twenty, years after iridectomy, respectively. Senile and glaucomatous changes causing loss of resistance in the tunics of the eye predispose to this accident.

BLESSIG (371, **Glaucoma and systemic disease**) notes that the vascular disturbance in glaucoma, alone, predisposes to congestive states and hyperæmia. This causal factor must be considered in constitutional treatment.

MELLER (373, **Cyclodialysis and its effect on tension**) reports his experiences with Heine's operation, which often gives good results but cannot be relied upon in all cases and does not replace iridectomy, except in high degrees of plus tension and advanced glaucoma which do not do well with iridectomy and in which this operation is difficult and dangerous. In this limited field cyclodialysis is a valuable procedure.

KRAUSS (374, **Cyclodialysis**) concludes from pathological studies that Heine's operation does not act by producing new filtration paths by means of choroidal detachment. Intraocular secretion is diminished and tension consequently falls, on account of an atrophy of the uveal tract due to the operation.

MARKLEITNER (375, **Traumatic cyclodialysis**) reports this condition without other complications as the result of an injury due to powder explosion. There was no discontinuity of tissues of cornea or sclera. The ciliary body was detached from the scleral ridge. In seven similar cases, iridodialysis, prolapse of iris or ciliary body, and choroidal detachment and rupture were noted as complications.

JUDIN (376, **Cyclodialysis: clinical and experimental study**) examined dogs' eyes post mortem after careful tonometric tests to determine the degree and duration of any diminution of tension following Heine's operation. He concludes that there is not only no filtration established with the suprachoroidal space, but that previous filtration becomes interfered with by adhesions as a result of the operation. He agrees with Krauss that cyclodialysis does not establish the communications claimed for it, as shown by these animal experiments and several microscopic examinations of human cyclodialyzed eyes. Good results are the exception, clinically, and complications are comparatively frequent. Diminution of tension is transitory and may be absent in human eyes. As a preliminary operation cyclodialysis may be of value in marked hypertony, synechia, or entire absence of anterior chamber, to diminish pressure temporarily and facilitate iridectomy.

STOELTING (379, **The normal operation in hydrophthalmus. Iridectomy or sclerotomy?**) finds that paracentesis of the anterior chamber is not sufficient. Sclerotomy, which he

performed in 16 cases, with good results in 10, is to be preferred.

CHALUPECKY (380, **Glaucoma and injury**) noted glaucomatous tension in 16 of 1200 cases of injury of the eye, but in only 6 was the causal relation undoubted. Acute traumatic glaucoma is very rare.

KUSCHEL (381, **Senile sclerosis of hydrostatic-regulation apparatus as predisposing cause of glaucoma**) notes that excessive traction and stretching of the anterior limiting layer is the main cause of the glaucomatous attack. This depends on senile sclerosis analogous to that of the systemic vessels and accompanied by changes in the muscles.

The patient described by VEASEY (384, **Glaucoma in the young**) was sixteen years of age when chronic glaucoma began in one eye, the dimness of vision first appearing during convalescence from typhoid fever. Vision was entirely lost in three years. Owing to development of pain the eye was enucleated. For seventeen years the other eye remained normal when glaucomatous symptoms made their appearance, but, as operation was refused, this eye also became blind.

ALLING.

XVIII.—SYMPATHETIC OPHTHALMIA.

385. PARISOTTI. **Experimental study on sympathetic ophthalmia.** *Soc. opht. franç.*, XXV., 1908.

386. LANS. **Sympathetic ophthalmia.** *Med. Weekblad*, xiv., p. 449.

387. KALT. **Peculiar case of sympathetic ophthalmia in open wound.** *Soc. opht. franç.*, XXV.

388. VALUDE, E. **Proper time for enucleation in sympathetic ophthalmia.** *Ibid.*

389. ABADIE, C. **Clinical and therapeutic study on sympathetic ophthalmia.** *Ann. d'ocul.*, CXXXIX., p. 409.

390. WIDMARK, J. **Salicylates in treatment of sympathetic ophthalmia.** *Mitth. a. d. Augen-Klin.*, Stockholm, No. 9, 1908.

391. GIFFORD, H. **Possible use of atoxyl in sympathetic ophthalmia, trachoma, and syphilis of the eye.** *Ophth. Record*, March, 1908.

392. HAMILTON, J. **Sympathetic ophthalmia.** *Royal London Hosp. Reps.*, January, 1908.

PARISOTTI (385, **Experimental study on sympathetic ophthalmia**) produced ciliary wounds in rabbits and noted a consensual irritation in both eyes, as indicated by an in-

creased albumin content of the aqueous. This is a constant preliminary stage of sympathetic ophthalmia and may be considered as a valuable premonitory symptom. Clinically it may be difficult to detect if it occurs alone.

VALUDE (388, **Proper time for enucleation in sympathetic ophthalmia**) thinks it is advisable to wait if the exciting eye still has some vision, but to enucleate at once if it is blind.

WIDMARK (390, **Salicylates in treatment of sympathetic ophthalmia**) gave from four to nine grams with good results in 8 of 12 cases, but notes that this form of treatment is apt to cause us to let the best time for operation go by.

GIFFORD (391, **Possible use of atoxyl in sympathetic ophthalmia, trachoma, and syphilis of the eye**) notes the affinity of atoxyl, an anilin compound of arsenic, for pathogenic protozoa. In a case of sympathetic ophthalmia he had an excellent result from this remedy although the interpretation of effects was clouded by the fact that salicylates and mercury were also given in large doses. For similar reasons he advises atoxyl in trachoma and syphilis of the eye. This drug has been known to cause blindness when given in large doses.

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HAMILTON (392, **Sympathetic ophthalmia**). Professor Fuchs ("Ueber sympathisierende Entzündung, nebst Bemerkungen über seröse traumatische Iritis," *Archiv für Ophthal.*, 1905, vol. cvi., part 2, page 365) examined a large amount of pathological material and selected 35 examples as being from subjects of sympathetic ophthalmia. With one exception in which *irritation* had been present, it was found on reference to the clinical histories that his diagnosis was correct. Janie Hamilton approached the research from the opposite standpoint. She examined microscopically eyes which she knew to be from cases of sympathetic ophthalmia.

The features which, according to Fuchs, are characteristic of sympathetic inflammation may be grouped under these heads:

I.—CELL FORMS.

(*a*) *Small mononuclear round cells—lymphocytes.* These correspond to the small round-celled infiltration found in all chronic inflammation, they are probably identical with the blood lymphocytes. These cells occur isolated or in nodes,

which can be seen under a low power, forming the so-called "patchy infiltration." They appear first in the region of the vessels. They increase in size and surround the vessel. Nodules coalesce and the inflammation becomes "diffuse."

(*b*) *Epithelioid cells.* These are a very constant feature; they are derived from the normal cells of the uveal stroma, from the endothelial linings of the vessels, and from the retinal pigment of the uvea.

(*c*) *Giant cells.* These are frequent, but not invariable. They are derived from epithelioid cells.

(*d*) *Mast cells* occur in many cases, but not in large numbers.

(*e*) *Polynuclear leucocytes.* These are rare, and probably indicate an accompanying non-sympathetic inflammation.

(*f*) *Russell's corpuscles* are very frequent; they are universal in tissue inflammation.

(*g*) *Plasma cells.* These are not insisted upon by Fuchs.

II.—The Distribution of the Infiltration.

The region essentially involved is the uvea—that is, the iris, ciliary body, and choroid.

The Iris. The infiltration is here insignificant. In 4% of Fuchs's cases it was hardly affected at all, in 11 equally with the rest of the uvea, in 3 (in all of which the iris was the entrance point of the original infection) it was chiefly involved.

The ciliary body is infected in every case, but sometimes only to a slight extent.

The choroid. It is here that the characteristic features are most marked. Of 35 cases Fuchs found that in 22 the choroid was intensely inflamed. It is never exempt. The changes are most marked in the posterior segment.

The first nodules appear in the stratum of large vessels. The chorio-capillaris on account of its elastic tissue escapes for a long time. The lamina vitrea is the next obstacle, and generally circumscribes the invasion. The retina therefore tends to escape.

III.—Resolution of the Infiltration.

The inflammation which is characteristic of sympathetic ophthalmia does not resolve by suppuration or caseation; it organizes in such a way that the uvea is transformed into a dense fibrous membrane.

IV.—Plastic Exudation.

This is mostly found in cases of long standing, but Fuchs believes that it indicates a mixed infection. The authoress here appends a useful tabular statement of the difference between sympathetic and ordinary septic infection.

Hamilton points out that Professor Fuchs has not given to the plasma all the attention it deserves. In all her cases she has made a quantitative estimation of the number of these cells, using Pappinheim's stain to differentiate them. A full description of this cell will be found in Mayou's *Hunterian Lectures on Conjunctivitis*, 1905. As the result of her research she concludes that Fuchs's description of the disease is correct. Any discrepancies are to be found in the sympathizing eye, which he did not include. His eyes were all "exciters." As regards sympathizing eyes, lymphocytes were abundant in four cases; giant cells were only once absent; plasma cells were exceptionally numerous; delicate fibreblasts were found in the exudates of several. Slight exudates were present over the iris and ciliary body. The infiltration seemed to be less rich in cells than is the case in the exciting eye. The appearance presented was that of tissues which were dissolving under the action of a powerful toxin.

In the exciting eyes the anatomical conditions described by Fuchs were almost invariably present. But the presence of the *plasma cell* is a well marked if not essential feature. Fuchs used Müller's fluid to fix his material and he had not the advantage of Pappenheim's specific stain. On the whole the number of plasma cells is greater in sympathetic than in non-sympathetic cases; they are more frequent in the sympathizing eyes than in exciters.

The summary of Professor Fuchs's paper is an excellent one, and will be most useful to those who cannot read German.

T. Harrison Butler.

To Illustrate Dr. De Schweinitz's Article on "Concerning a Form of Retinal Disease, with Extensive Exudation."

Retinal disease with extensive exudation.

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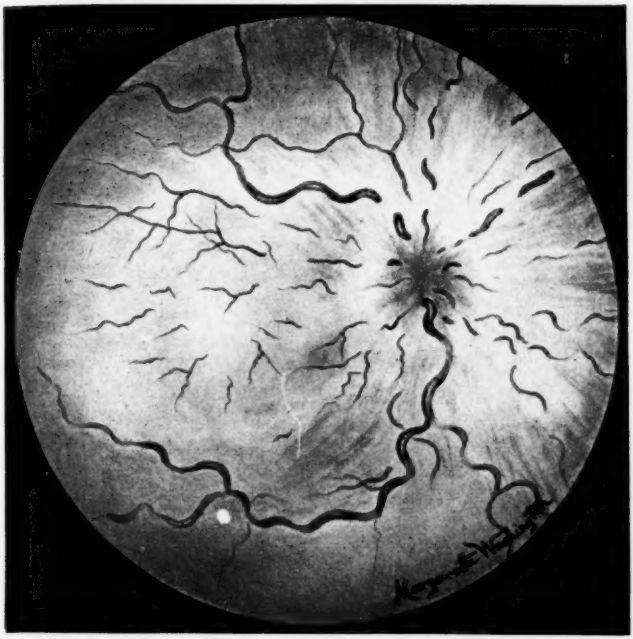
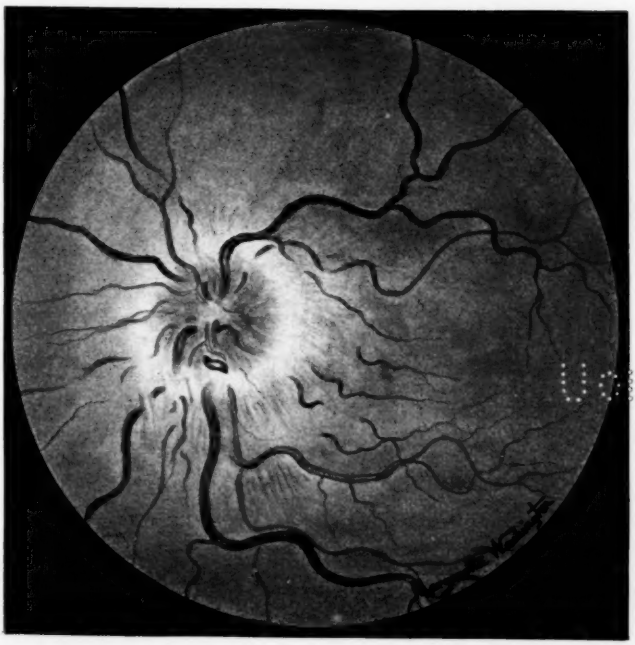
To Illustrate Dr. Posey's Article on "Neuroretinitis in Chlorosis."





A case of intense neuroretinitis in chlorosis.

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TO ILLUSTRATE DR. SHUMWAY'S ARTICLE ON "METASTATIC CARCINOMA OF THE ORBIT."

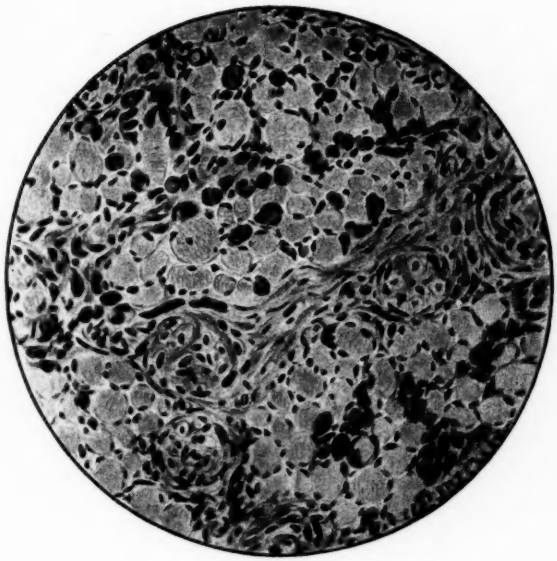

FIG. 1.—Cross section of rectus muscle, showing infiltrating carcinoma cells and several nerve bundles

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